

**Prof. Dr. Ojha Sir's Facebook post about different topics....**

**From August 2012 to October 2017**

**PREFACE-:**

*Today everyone is connected with each other by the social media and facebook is one of the famous social networking Site. Posts on facebook make the people aware about the recent updates. It is a best platform to share our thoughts and knowledge. Keeping this in mind, Prof. Dr. S. N. Ojha sir has been posting different study related posts on various topics, since last 5 years.*

*Dr. Ojha sir is having large numbers of students from all over India and facebook is the best way to reach them. With his unique thoughts and concepts, he has posted wide range of topics including Cardiology, Oncology, Endocrinology, Aavarana, etc., both with Ayurved and Modern approach.*

*The knowledge which he is sharing with us on facebook is so precious that it should not be limited mere as a post for receiving 'Likes', instead, it must be preserve with us throughout the life. Hence, I have just made a small attempt to collect his all study related posts and compile in proper sequence so that it will become useful source of knowledge for the students and practioners. It will also help those students who have newly joined with him or those who have missed his previous posts. I am very pleased to present this collection and compilation of different study related posts on Facebook, posted by Prof. Dr. S. N. Ojha, from the August 2012 to October 2017.*

*Thank you!*

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## **Aama , autoimmune phenomennon , and ayurveda.→**

Certain biomarkers like immune complexes can be considered as aama and they are measurable..Oxidized LDL - cholesterol can be correlated with saama meda..Ch.chi.15 in reference to ajeerna , acharya charak is very clear to demonstrate aama and its related disorders.. autoantibodies are best examples of circulating aama

There is autoantibody to islet cells of langerhans causing type 1 DM , aama in samaana sthaana and manifesting madhumeha.. We should evaluate immunomodulator effect of mustaa like drugs in clinical / experimental trial..

Mustaa can grow at any place ,shows its strongest behaviour to survive..CRP is acute phase reactant , can be considered as rasa/rakta gata aama marker..triglycerides is major culprit in chronic complications of DM , biomarker for saama meda /kapha , responsible for atherogenesis...For such purpose our choice is katu and tikta dravya enriched in vaayu agni and aakaasha mahaabhoota helping in countering the saamata ..Rasaadibhihcha samsrishtam kuryaat rogaan rasaadijaan ch.chi.15/49...Antibody to gastric parietal cells →atrophic gastritis → amlapitta.. paandu...Ghora anna visha / environmental factors , especially diet components are triggering factors in genetic predisposed persons to induce auto immunity..The identification of aama , cross reaction due to molecular mimicry and self to non self reaction are very crucial in auto immunity.. Identification of aama by vaayu , molecular mimicry by kapha and self to non self reaction by pitta and eventually initiation of auto immune induced disease process is concerned with tridosha...amrita , pippali , patola , kiraatatikta , shunthi , kumaari ,ashwagandhaa , shataavari , balaa , etc are effective in autoimmune disorders.



6 April 2016

## Antiphospholipid Syndrome (APS)

**Antiphospholipid Syndrome (APS)** is an autoimmune disease which can cause frequent clotting in arteries and veins and/or miscarriages. The clotting results from the presence of proteins in the blood called anti-phospholipid autoantibodies (commonly called aPL) formed against the person's own tissues. Tikta and katu rasaatmaka dravya should be used , sroto shuddhi , prashashta dhaatu nirmiti are concerned.. Kaphaanubandhi Raktapitta is reason of frequent clotting.. Kapha and aama , later srotorodha and hence vaata prakopa are present in APS.. Treatment plan as per aamavaata and kaphaanubandhi Raktapitta.. Kansa haritaki , mahasudarshan churna , vardhamaana pippali ( if tolerable ) , kamala kshaara & parawata shakrit ( as clotlytics) virechan , and vaitarna basti ( short course) , to break autoimmune phenomena .

## The relationship between āma , oxidative stress and atherosclerosis..

### Acharya charak , acharya chakrapani and my approach ;

Acharya on Ca. Ci. 15/ 42-43 has explained snigdha, uṣṇa, laghu, śīta, laṅghana, adhyāśan, vēgavidhāraṇ prajāgaran can cause agnidushti whereas in Ca. Vi. 5/9 bhaya etc manasik bhava has been mentioned which causes indigestion although proper food and quantitative food is taken.

Drava, śuṣka, dadhi, kṣīra, grāmya, ānūpa, udaka, āmiṣa, piṣṭa, vyāpanna, madya, ātdivāsvapna are the causes mentioned in sthaulya and the same mentioned in rasavaha srotodushti. Further Sushruta has metioned sthaulya due to rasa dushti. Thus a process of agni dushti followed apachit rasa dhatu formation followed by formation of sama dosa and sama dhatu.

It may be recalled that rasa mentioned here is rudhiradi i.e rakta, mansa, meda, asthi etc in the form of drava rupa.

Ama has been explained as one of the essential factor in pathogenesis of various diseases. Dosha, dusya, srotodushti, agni and ama play a collaborative role in the samprapti of a vyadhi. Charak in Trividha Kukshiya Adhyaya has explained that food and human behavior contribute to formation of ama. The ama further causes vitiation of one or all of the three doshas which further contribute to eksthan or sarvadaihik vyadhis.

Acharya Vagbhata was the one who defined Ama as a entity which is caused by durbala or alpa bala of agni whereby aadya dhatu i.e. none other than rasa dhatu is formed in apachit swaroop. The dushit rasa which when reaches the amashaya it is known as ama<sup>2</sup>. Sarvangasundarkar while commenting on the word dushtam says vatadianusayitam i.e. alongwith vatadi dosha<sup>3</sup>. The apachit rasadhatu due to agnimandhya alongwith vatadi dosha contribute to the formation of ama.

Vijayrakshit in Madhukoshatika on jwara adhyaya explains that daurbalyata of jatharagni leads to formation of avipakwa rasa which is known as ama which is responsible for sarvadaihika dosha prakopa<sup>4</sup>.

JATHARANALA DAURBALYAT AVIPAKWASTU YO RASA II

SA AMA SADNNYAKO DEHE SARVA DOSHA PRAKOPANA II MADHUKOSHA

By above explanation one can limit his knowledge to intermediate metabolites because avipakwa aahar rasa is one of the steps of digestion. It is the reason why Vagbhat did not restrain his definition only to agnimandhya but also said that anyonya murchana of dushit dosha can also cause ama similar to kodrava (*Paspalum scrobiculatum* Linn.)<sup>5</sup>. *Paspalum ergot* is a fungal disease that kodo millet is susceptible to. Hardened masses of this fungus will grow in place of the millet grain, called sclerotia. These compact fungi growths contain a chemical compound that is poisonous to humans and livestock if consumed, and potentially fatal.

Madhukoshkara has kept a similar approach and has classified samata of two types “eka rasasya apara doshasya” in reference to explanation of sama jwara lakshana<sup>6</sup>. {Madhav nidan madhukosha tika 2/65}.

SO ANNAJO RASA ITI AAMA:, ANNARASASYAEVA APAKWASYA TANTRANTARE  
AMAVYAPADESHAT II Madhukosha vyakhya

AAHARASYA RASA SHESHO YO NA PAKWO AGNI LAGHAWAT II

SA MULAM SARVAROGANAM AMAM ITYABHIDIYATE II

Madhukosha vyakhya 25/5

Apakwa aahar rasa is called as ama and it is mula karan for sarvarog.

In context of amavata Vijayrakshit has quoted other acharyas that ama is apachit annarasa, prathamam doshadushtikar or even malasanchaya<sup>7</sup>.

AMA ANNARASAM KECHIT KECHITU MALASANCHAYAM II

PRATHAMAM DOSHADUSHTIM CHA KECHID AMAM PRACHAKSHATE II

Therefore while describing the treatment Acharyas have considered 3 condition of ama viz.

SARVADEHA PRAVISRUTHAN SAMAN DOSHAN- Apachit annarasa

LINAN DOSHAN- prathamam doshadushtikar

ASHRAYASYA HI – mala sanchaya

Charak in trividhakukshiya adhyaya has explained two types of ama pradosha<sup>8</sup>. Chakrapaani commenting on the word Amapradosha has explained two different conditions:

AMA APAKWAM SA DUSTA DOSHA SAMPARKAT SHARIRAM DUSAYATI

AMA PRADUSAYATI ITI ATRA KARMA KATRUTWE ACHA DUSTAM BHAVATI ITI  
ARTHA:

Therefore it means that either ama is vitiated by dusta dosha causing sharir dushti or the dushit apakwa rasa itself has the capacity to cause the disease by vitiating the doshas<sup>9</sup>.

In this context one needs to understand the concept of dosha in relation to ama.

Dosha has a wide scope in comparison to the modern aspect in relation to sthan and karma. Considering the gastrointestinal tract previously kapha has been compared with mucus secretion, pitta with digestive secretion and vata with neural activity but a recent research in modern science related to free radicals and oxidation process, ROS, needs to be understood. This may be vata dharma dravya or pitta dharma dravya or kapha dharma dravya.

Free radicals are molecules with unpaired electron in their outer orbit. Free radicals have very important role in origin of life and biological evolution, leaving beneficial effects on the organisms. Many of these are necessary for life, such as the intracellular killing of bacteria by phagocytic cells such as granulocytes and macrophages. Researchers have also implicated free radicals in certain cell signaling processes,<sup>[7]</sup> known as redox signaling.

Oxygen radicals are involved in many biochemical activities of cells such as signal transduction, gene transcription and regulation of soluble guanylate cyclase activity. Nitric oxide (NO) is an important signaling molecule that essentially regulates the relaxation and proliferation of vascular smooth muscle cells, leukocytes adhesion, platelets aggregation, angiogenesis, thrombosis, vascular tone and hemodynamics

The two most important oxygen-centered free radicals are superoxide and hydroxyl radical. They derive from molecular oxygen under reducing conditions.



However, because of their reactivity, these same free radicals can participate in unwanted side reactions resulting in cell damage. Overproduction of free radicals can cause oxidative damage to biomolecules, (lipids, proteins, DNA), eventually leading to many chronic diseases such as atherosclerosis, cancer, diabetics, rheumatoid arthritis, post-ischemic perfusion injury, myocardial infarction, cardiovascular diseases, chronic inflammation, stroke and septic shock, aging and other degenerative diseases in humans . Excess NO is cytotoxic either by combining with tyrosine that is essential for catalytic function of enzyme ribonucleoside diphosphate reductase or by forming ONOO<sup>-</sup>. Excess vascular O<sub>2</sub> production could contribute to hypertension and vasospasm [39, 49, 60]. Some of the symptoms of aging such as atherosclerosis are also attributed to free-radical induced oxidation of cholesterol to 7-ketocholesterol.

Human body produce oxygen free radicals and other reactive oxygen species as by products through numerous physiological and biochemical processes. Oxygen related free radicals (superoxide and hydroxyl radicals) and reactive species (hydrogen peroxide, nitric oxide, peroxy nitrile and hypochlorous acid), are produced in the body, primarily as a result of aerobic metabolism [34, 66]. Some free radicals arise normally during metabolism. Sometimes the body immune systems cells purposefully create them to neutralize viruses and bacteria. However, environmental factors such as pollution, radiation, cigarette smoke and herbicides can also spawn free radicals.

Free radicals attack important macromolecules leading to cell damage and homeostatic disruption. Targets of free radicals include all kinds of molecules in the body. Among them, lipids, nucleic acids, and proteins are the major targets.

Normally, the body can handle free radicals, but if antioxidants are unavailable, or if the free-radical production becomes excessive, damage can occur. Of particular importance is that free radical damage accumulates with age. Cancer and atherosclerosis, two major causes of death, are salient “free radical” diseases.

Secondly, Reactive Oxygen Species or ROS are species such as superoxide, hydrogen peroxide, and hydroxyl radical, commonly associated with cell damage. ROS form as a natural by-product of the normal metabolism of oxygen and have important roles in cell signaling.

ROS attack the polyunsaturated fatty acid, linoleic acid, to form a series of 13-Hydroxyoctadecadienoic acid and 9-Hydroxyoctadecadienoic acid products that serve as signaling molecules that may trigger responses that counter the tissue injury which caused their formation. ROS attacks other polyunsaturated fatty acids, e.g. arachidonic acid and docosahexaenoic acid, to produce a similar series of signaling products.

ROS are particularly active in the brain and neuronal tissue as the excitatory amino acids and neurotransmitters, whose metabolism is factory of ROS, which are unique to the brain and serve as sources of oxidative stress. ROS attack glial cells and neurons, which are post-mitotic cells and therefore, they are particularly sensitive to free radicals, leading to neuronal damage . It has been reported that deleterious effects of ROS on human cells may end in oxidative injury leading to programmed cell death i.e. apoptosis.

Even Leukocytes and other phagocyte destroy bacteria, parasites and virus-infected cells with NO, O<sub>2</sub>, H<sub>2</sub>O<sub>2</sub>, and OCl, those are powerful oxidants and protect humans from infection. However, they cause oxidative damage and mutation to DNA and participate in the carcinogenic process if unchecked.

Thus it can be understood that Free radicals, ROS, Leukocytes are helpful (dharan of sharir) when in normal and the same become enemy when vitiated. The aetiological factors mentioned in urustambha causes metabolic derangement (agni mandhya) thereby leading to vitiation of this factors (dosha) and the same leads to various disorders.

For eg. Madhya has a role in urustambha, Chronic alcoholic myopathy affects up to two-thirds of all alcohol misusers and is characterized by selective atrophy of

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Type II (glycolytic, fast-twitch, anaerobic) fibers. In contrast, the Type I fibers (oxidative, slow-twitch, aerobic) are relatively protected. Alcohol increases the concentration of cholesterol hydroperoxides (sama meda) and malondialdehyde-protein adducts, though protein-carbonyl concentration levels do not appear to be overtly increased and may actually decrease in some studies. In alcoholics, plasma concentrations of alpha-tocopherol may be reduced in myopathic patients. However, alpha-tocopherol supplementation has failed to prevent either the loss of skeletal muscle protein or the reductions in protein synthesis in alcohol-dosed animals. The evidence for increased oxidative stress in alcohol-exposed skeletal muscle is thus inconsistent. Further work into the role of ROS in alcoholic myopathy is clearly warranted.

(Free radicals in alcoholic myopathy: indices of damage and preventive studies. Preedy VR1, Adachi J, Asano M, Koll M, Mantle D, Niemela O, Parkkila S, Paice AG, Peters T, Rajendram R, Seitz H, Ueno Y, Worrall S.)

It is seen that free radicals are formed during muscular ischemia leading to further damage of muscle. Atherosclerosis leads to muscular damage. Atherosclerosis, one contributing factor is the lipids (sama meda). Studies on atherosclerosis reveal the probability that the disease may be due to free radical reactions involving diet-derived lipids in the arterial wall and serum to yield peroxides and other substances. These compounds induce endothelial cell injury and produce changes in the arterial walls.

A role of oxidative stress has been postulated in many conditions, including atherosclerosis, inflammatory condition, certain cancers, and the process of aging. Oxidative stress is now thought to make a significant contribution to all inflammatory diseases (arthritis, vasculitis, glomerulonephritis, lupus erythematosus, adult respiratory diseases syndrome), ischemic diseases (heart diseases, stroke, intestinal ischemia), hemochromatosis, acquired immunodeficiency syndrome, emphysema, organ transplantation, gastric ulcers, hypertension and preeclampsia, neurological disorder (Alzheimer's disease,



Parkinson's disease, muscular dystrophy), alcoholism, smoking-related diseases, and many others. An excess of oxidative stress can lead to the oxidation of lipids and proteins, which is associated with changes in their structure and functions.

Atherosclerosis is initiated by inflammatory processes in the endothelial cells of the vessel wall associated with retained low-density lipoprotein (LDL) particles. This retention may be a cause, an effect, or both, of the underlying inflammatory process. (kha vaigunya caused by the hetu sevan in specific srotas) Lipoproteins in the blood vary in size. Some data suggests that small dense LDL (sdLDL) particles are more prone to pass between the endothelial cells, going behind the cellular monolayer of endothelium. LDL particles and their content are susceptible to oxidation by free radicals, (dosha as discussed above) and the risk is higher while the particles are in the wall (sira sthita meda) than while in the bloodstream.

Once inside the vessel wall, LDL particles can become more prone to oxidation. Endothelial cells respond by attracting monocyte white blood cells, causing them to leave the blood stream, penetrate into the arterial walls and transform into macrophages. (dosha dusyanti). The macrophages' ingestion of oxidized LDL particles triggers a cascade of immune responses which over time can produce an atheroma if HDL removal of fats from the macrophages does not keep up. The immune system's specialized white blood cells (macrophages and T-lymphocytes) absorb the oxidized LDL, forming specialized foam cells. If these foam cells are not able to process the oxidized LDL and recruit HDL particles to remove the fats, they grow and eventually rupture, leaving behind cellular membrane remnants, oxidized materials, and fats (including cholesterol) in the artery wall. This attracts more white blood cells, resulting in a snowballing progression (samata increased but this samata is not due to agnimandhya alone, it is due to dosha conglomeration the second definition of samata given by Vagbhata) that continues the cycle, inflaming the artery. The presence of the plaque induces the muscle cells of the blood vessel to stretch, compensating for the additional bulk,



and the endothelial lining thickens, increasing the separation between the plaque and lumen. This somewhat offsets the narrowing caused by the growth of the plaque, but it causes the wall to stiffen and become less compliant to stretching with each heart beat.

### Mechanism

Atherogenesis is the developmental process of atheromatous plaques. It is characterized by a remodeling of arteries leading to subendothelial accumulation of fatty substances called plaques. The buildup of an atheromatous plaque is a slow process, developed over a period of several years through a complex series of cellular events occurring within the arterial wall, and in response to a variety of local vascular circulating factors. One recent hypothesis suggests that, for unknown reasons, leukocytes, such as monocytes or basophils, begin to attack the endothelium of the artery lumen in cardiac muscle. The ensuing inflammation leads to formation of atheromatous plaques in the arterial tunica intima, a region of the vessel wall located between the endothelium and the tunica media. The bulk of these lesions is made of excess fat, collagen, and elastin. At first, as the plaques grow, only wall thickening occurs without any narrowing. Stenosis is a late event, which may never occur and is often the result of repeated plaque rupture and healing responses, not just the atherosclerotic process by itself.

In effect, the muscular portion of the artery wall forms small aneurysms just large enough to hold the atheroma that are present. The muscular portion of artery walls usually remain strong, even after they have remodeled to compensate for the atheromatous plaques.

However, atheromas within the vessel wall are soft and fragile with little elasticity. Arteries constantly expand and contract with each heartbeat, i.e., the pulse. In addition, the calcification deposits between the outer portion of the atheroma and the muscular wall, as they progress, lead to a loss of elasticity and stiffening of the artery as a whole.

If the fibrous cap separating a soft atheroma from the bloodstream within the artery ruptures, tissue fragments are exposed and released. These tissue fragments are very clot-promoting, containing collagen and tissue factor; they activate platelets and activate the system of coagulation. The result is the formation of a thrombus (blood clot) overlying the atheroma, which obstructs blood flow acutely. With the obstruction of blood flow, downstream tissues are starved of oxygen and nutrients. If this is the myocardium (heart muscle) angina (cardiac chest pain) or myocardial infarction (heart attack) develops and if in skeletal muscles the muscle ischaemia takes place reducing the muscle strength and leading to myopathy.

#### Risk factors

Various anatomic and physiological risk factors for atherosclerosis are known. But considering sthaulya as mentioned in asthonditiya adhyaya, madhumeha as mentioned in Ca. Su. 17, urustambha and meda sa avrita vata/ upastambhita vata prakopa following risk factors are common. It may be noted that in all the above mentioned disease meda and vata is common.

Hyperlipidemia, hypertension and cigarette smoking together increases the risk seven times.

Diabetes or impaired glucose tolerance (IGT) +

Dyslipoproteinemia (unhealthy patterns of serum proteins carrying fats & cholesterol): +

High serum concentration of low-density lipoprotein (LDL, "bad if elevated concentrations and small"), and / or very low density lipoprotein (VLDL) particles, i.e., "lipoprotein subclass analysis"

Low serum concentration of functioning high density lipoprotein (HDL "protective if large and high enough" particles), i.e., "lipoprotein subclass analysis"

An LDL:HDL ratio greater than 3:1

Obesity.(in particular central obesity, also referred to as abdominal or male-type obesity) +

High intake of saturated fat (may raise total and LDL cholesterol.

Intake of trans fat (may raise total and LDL cholesterol while lowering HDL cholesterol).

High carbohydrate intake.

Elevated serum levels of triglycerides

संचयं च प्रकोपं च प्रसरं स्थानसंश्रयम् । व्यक्तिं भेदं च यो वेत्ति दोषाणां  
स भवेत् भिषक् ॥ सु .36/21 .सू.

Acharya Sushruta has described the concept of Kriyakala which seeks to explain the incidence of vranas, in terms of dosha disturbances. Vrana in modern parlance may be described as inflammatory processes, which may lead on to suppuration and ulceration. The inflammatory processes are stated to follow a distinct pattern of evolutive phases which are described as .Kriyakala

Sanchaya, Prakopa, Prasar, Sthansanshraya, Vyakti and Bheda are the six kriyakala. All the diseases undergo this process of evolutory changes. The utility of the same may be understood in context of fever.

**Sanchaya:** It is the inceptive phase of disease when dosha are stated to have accumulated and stagnated in its own place. In case of fever it is the pyrogen which may be exogenous (bacterial substance lipopolysaccharide (LPS) present on bacterial cell wall) or endogenous (cytokines, Interleukin-1 and Interleukin-6 etc). These pyrogens enter the body and activate the immune cells (antigen presenting cell) for the formation of cytokines and other factors or due to endogenous cause too activation of immune system takes place.

**Prakopa:** It is phase wherein accumulated and stagnated dosha tend to become swollen and excited in its own place. Exogenous factors contain immunological protein called lipo-polysaccharide binding protein (LBP) which binds to LPS. The LBP-LPS complex then binds to the CD14 receptors of a nearby macrophage. It causes synthesis and release of various endogenous cytokines factors such as IL-1, IL-6, Tumour Necrosing Factor alpha (TNF $\alpha$ ).

**Prasara:** It means to spread which generally takes place with help of vata and rakta. The excited and swollen dosha are stated to spread over and extend to



other parts of the body. These cytokine factors are released into general circulation, where they migrate to the circumventricular organs of the brain due to easier absorption caused by the blood–brain barrier's reduced filtration action there. The cytokine factors then bind with endothelial receptors on vessel walls, or interact with local microglial cells. When these cytokine factors bind, the arachidonic acid pathway is then activated. Prostaglandin E<sub>2</sub> (PGE<sub>2</sub>) is released which is mediated by the enzymes phospholipase A<sub>2</sub> (PLA<sub>2</sub>), cyclooxygenase-2 (COX-2), and prostaglandin E<sub>2</sub> synthase.

**Sthanasansraya:** It is prodromal phase or the phase of purvarupa wherein disease is yet to be manifested fully. The excited dosha having extended to other parts of the body become localized and it marks the beginning of specific diseases pertaining to those sthan/ structures. PGE<sub>2</sub> is the ultimate mediator of the febrile response. PGE<sub>2</sub> acts on neurons in the preoptic area (POA) through the prostaglandin E receptor 3 (EP3). EP3-expressing neurons in the POA innervate the dorsomedial hypothalamus (DMH), the rostral raphe pallidus nucleus in the medulla oblongata (rRPa), and the paraventricular nucleus (PVN) of the hypothalamus. Fever signals sent to the DMH and rRPa lead to stimulation of the sympathetic output system, which evokes non-shivering thermogenesis to produce body heat and skin vasoconstriction to decrease heat loss from the body surface. It is presumed that the innervation from the POA to the PVN mediates the neuroendocrine effects of fever through the pathway involving pituitary gland and various endocrine organs.

**Vyakti:** This stage may be stated to be that of manifestation of the fully developed disease- the resultant dosha dushya samurchana. In case of fever the brain ultimately orchestrates heat effector mechanisms via the autonomic nervous system. It causes increased heat production by increased muscle tone, shivering and hormones like epinephrine (adrenaline) and also prevents heat loss by way of vasoconstriction.

**Bheda:** It is the stage in which the disease may become sub-acute and chronic or incurable. Different types or variant of disease gets manifested. Signs like increased blood pressure, neck stiffness, headache, giddiness, unconsciousness etc are seen in this phase.

The utility of this shatkriyakala is to enable the treating physician to recognize the disturbances in its early formative stages and to enable to take necessary steps in time, to correct and eliminate the offending factors before they have caused sufficient damage.

### Shat-kriyakala - modern time review:

Acharya Sushruta has described the concept of Kriyakala which seeks to explain the incident of vrana in terms of dosha disturbance. Vrana in modern parlance may be described as inflammatory process which may lead on to suppuration and ulceration. The concept of Kriyakala describes the mode and stages of the development of diseases. A good understanding of Kriyakala is very essential for early diagnosis, prognosis and for adopting preventive and curative measurement.

The term Kriyakala means the time of action. Kala or time in this context signifies the avastha or stage of the process of diseases.

Kalo hi nityaga avasthika; tatra avasthika vikaram apekshate ||

Ca.Vi.1/229(6)

Kriya or action refer to the resort to measure-aushadha, ahara and charya-with a view to eliminate and correct the doshic disturbance.

Kriyakala therefore, means the (early) recognition of the avastha or the stage of the process of disease and the resort to appropriate measures to correct the same.

As described above shatkriyakala are explained in vrana prashna adhyaya where Dalhan the commentator clarifies vrana in this content is not wound but vatadi humor (dosha) which themselves are cause for dehotpatti (responsible for structural and functional activity of body). They are the one who maintain normalcy. They physiologically go through the phase of chaya prakopa and prashama. This 3 step process is essential for sharira dharan.

The same dosha when get vitiated are cause for destruction of sharir.

Ta eva cha vyapanna pralaya hetava ||

Su.Su.21/3

Destruction (vyapanna) in case of sharir refers to vikruti or disease which undergoes evolution in 6 phase viz chaya, prakopa, prasara, sthanasansraya, vyakti and bhedha.

Inflammation is the local physiological response to tissue injury. It is not in itself, a disease, but is usually a manifestation of disease. Inflammation may have beneficial effect such as the destruction of invading micro-organisms and the walling-off of an abscess cavity to prevent spread of infection. However, may also produce diseases; for example, an abscess in the brain wound act as space occupying lesion compressing vital surrounding structure, or fibrosis resulting from chronic inflammation may destroy tissue permanently.

Inflammation is a protective response that involves immune cells, blood vessels and molecular mediator. It is one among the reason why Acharya Sushruta in this context has accepted rakta (shonita) as fourth dosha. Inflammation is a generic response, and therefore it is considered as a mechanism of innate immunity as compared to adopted immunity specific for each pathogen/hetu. Thus one hetu can cause many diseases and many hetu can cause one disease.

Sanchayam ca prakopam ca prasaram sthana sansrayam ||

Vyaktim bheda ca yo vetti doshanam sa bhaveda bhisaka ||

**SANCHAYA:** It is the first phase of shat kriyakala; it is the stage of accumulation or the stage which represents the inceptive phase of the disease wherein the dosha are stated to have accumulated and stagnated in its own place (Dosha sthanani yesu sanchiyate || Su. Su. 21/28), instead of freely circulating as in its normal avastha or phase.

Dosha in this condition are in compact form (Samhati rupa vridhi chaya || Dalhan Su. Su. 21/18). Samhata or compactness can be understood by symptom of vata dosha, vata dosha chaya which is manifested as stabdha purna kostha i.e. stabdha kostha (sense of dullness in abdomen/ sense of reduced intestinal motility) and purna kostha (sense of fullness/ sense of heaviness in abdomen). To fill up the space is normal function of vata which is manifested in excess in sanchaya avastha.

Pitta chaya is manifested with yellow tinge (pittavabhasata). Yellow is normal colour of pitta which is manifested as excess.

Kapha manifest as low body temperature/ reduce temperature (manda ushmata), heaviness of a part or full body (anga gaurav) and languor (alasya). These symptoms are seen in sthan (seat) where chaya rupa vridhi is seen.

The process of acute inflammation is initiated by resident immune cells (sthanik dosha) already present in the involve tissue mainly resident macrophages, dendritic cells, histiocytes, Kupffer cells and mastocytes. Receptors named pattern recognition receptors (PRRs) recognize generic molecules that are broadly shared by pathogens but distinguishable from host molecules, collectively referred to as pathogen associated molecular pattern (PAMPs). Thus cells undergo activation and recognize non self and mechanism of opposition is initiated which is manifested as pradvesho vridhi hetusu i.e. aversion towards similar and attraction towards contraries.



In case of fever it is the pyrogen which may be exogenous (bacterial substance lipopolysaccharide (LPS) present on bacterial cell wall) or endogenous (cytokines, Interleukin-1 and Interleukin-6 etc). These pyrogens enter the body and activate the immune cells (antigen presenting cell) for the formation of cytokines and other factors or due to endogenous cause too activation of immune system takes place.

Atherosclerosis, formally considered a bland lipid storage disease, actually involves an ongoing inflammatory response. Recent advances in basic science have established a fundamental role for inflammation in mediating all stages of this disease from initiation through progression and ultimately, the thrombotic complications of atherosclerosis. Oxidised levels of LDL, increased level of VLDL are the initiation or triggering factor of atherogenesis.

Sanchaya is the early initiation of marker C-reactive Protein which prospectively defines risk of atherosclerotic complications, thus adding to prognostic information provided by traditional risk factors.

Thus new insights into inflammation in atherosclerosis not only increase understanding of disease but also have practical clinical application in risk stratification and targeting of therapy for this scourge of growing worldwide importance rightly said by Dalhan as prathama kriyakala aadya karmavasara.

**PRAKOPA:** In this stage dosha gets vitiated or aggravated or the dosha previously accumulated/ stagnated get swollen and excited. Vilayan rupa vridhi prakopa (Dalhan). Vilayana here means bonding (samhata) is loss and dosha gets released.

In case of acute inflammation release of inflammatory mediators responsible for the clinical signs of inflammation takes place.

In case of fever Exogenous factors contain immunological protein called lipopolysaccharide binding protein (LBP) which binds to LPS. The LBP-LPS complex then binds to the CD14 receptors of a nearby macrophage. It causes synthesis and

release of various endogenous cytokines factors such as IL-1, IL-6, Tumour Necrosing Factor alpha (TNF $\alpha$ ).

In atherosclerosis C- Reactive Protein is elevated and noted in the blood test. Increased level of LDL, VLDL and intermediate lipoproteins activate inflammatory functions of vascular endothelial cells. During atherogenesis, inflammatory cells (eg, monocyte-derived macrophages) accumulate in arteries, releasing growth factors/cytokines (eg, platelet-derived growth factor [PDGF], transforming growth factor-beta [TGF- $\beta$ ], granulocyte-macrophage colony-stimulating factor). Whereas PDGF may stimulate cholesteryl ester (CE) hydrolysis in cells, TGF- $\beta$  appears to cause a decrease in lysosomal CE hydrolysis. The latter could lead to a transient reduction in intracellular free cholesterol.

In case of Allergens pre-sensitized mast cells respond by degranulating, releasing vasoactive chemicals such as histamine.

Clinical knowledge of Acharyas is saluted by significant and pertinent observation made by Sushruta to rakta as the medium (or substrate) for the spread or dissemination of the morbid factors of the disease. The aggravation of the dosha goes together with the disturbed or agitated state of rakta.

Yasmad rakta.....

Dalhan says alone dosha are unable to get prakopita whereas they are always dependent (paratantra) on rakta. Therefore Acharya have mentioned vata, pitta and kapha dushita rakta. Modern Science too explains release of inflammatory mediators in blood. Vasodilation and its resulting blood flow cause the redness (rubor) and increased heat (calor), (paridaha).

Acute inflammation is an immune-vascular response to an inflammatory stimulus. Vascular response is compared with rakta prakopa and cellular/ immune response to vata, pitta and kapha.

Similarly upon contact with PAMPs, tissue macrophages and mastocytes release vasoactive amines such as histamine and serotonin, as well as eicosanoids such as prostaglandin E2 and leukotriene B4 to remodel the local vasculature. Macrophages and endothelial cells release nitric oxide. These mediators vasodilate and permeabilize the blood vessels, which results in the net distribution of blood plasma from the vessel into the tissue space.

**PRASARA:** The third phase signifies to spread which generally takes place with help of vata and rakta. Dosha are stated to spread over and extend to other parts of the body.

TESHAM VAYUGATIMATVAT PRASARAN HETU SATYA API ACHAITANYA I

RAJASCA PRAVARTANA SARVABHAVANAM II

The biomotor or motive force which keeps the rakta moving all over the body, through its own channels-srotas- is vata.

The doshas which have become prakupita expand and overflow the limits of their respective locations. This is explained with two analogues viz the overflow which occurs during the process of fermentation in which ferments rises acquiring new and unseen qualities and the later analogy refers to the overflowing in water dam due to an increased accumulation of water in it, resulting in the two sides of the dam being connected into one vast and continuous sheet of water. It explains the various pressure gradients which enable the vimargagaman of inflammatory mediators from vascular tract into another tissue space, organ system or tract. Pressure gradient cause permeability of srotas/ channels and due to unknown reason dosha do the dusti of rakta and prasar of dushit rakta takes place through 15 different ways as mentioned by Acharya Sushruta.

In case of fever the cytokine factors are released into general circulation, where they migrate to the circumventricular organs of the brain due to easier absorption caused by the blood-brain barrier's reduced filtration action there. The cytokine



factors then bind with endothelial receptors on vessel walls, or interact with local microglial cells. When these cytokine factors bind, the arachidonic acid pathway is then activated. Prostaglandin E2 (PGE2) is released which is mediated by the enzymes phospholipase A2 (PLA2), cyclooxygenase-2 (COX-2), and prostaglandin E2 synthase.

In acute inflammation the inflammatory mediators molecules alter the blood vessels to permit the migration of leukocytes, mainly neutrophils and macrophages, outside of the blood vessels (extravasation) into the tissue. Vimargagaman as explained by Sushruta. The neutrophils migrate along a chemotactic gradient created by the local cells.

Increased permeability of blood vessels results in the net distribution of blood plasma from the vessel into the tissue space.

Vasodilation occurs first at the arteriole level (prakopa) progressing to the capillary level, and brings about an increase in the amount of blood present causing the redness and heat of inflammation. Thus paridaha symptom is present in Prakopa and prasar stage of shatkriyakala.

In allergy vasoactive chemicals like histamine propagate an excessive inflammatory response characterized by blood vessel dilation and cytokine release into the blood which move along with blood.

Acharya Sushruta analogues that the manner in which rain loaded clouds downpour in specific area where they are taken with help of wind similarly dosha whether permeating the entire body or a part of it- ardha sharira or become confined to a particular part or a member of the body, may give rise to disease in the site of their transportation.

Further Sushruta has also explained how sometime simple cause trigger exacerbated symptoms of disease. Sushruta says prakupita doshas when not sufficiently excited may remain quiescent, coating (lina dosha) the internal



pathways- margas- of the body and exacerbate to cause disease, when they are subsequently excited by appropriate exciting factors.

The above quiescence can be easily understood when patient says previous night he had egg and from next day he started with bloody stools with increased frequency which was later on diagnosed as Ulcerative Colitis, an inflammatory bowel disease. Here egg is exciting factor whereas in patient body the dosha/ inflammatory mediators were already prakopita and waiting for exciting causes.

Allergic rhinitis, urticarial etc are example of lina dosha wherein vascular response secrete histamine which excitingly stimulates cellular immunity to show up sudden (achaya purvak) symptoms.

**STHANASANSRAYA:** It is prodromal phase or the phase of purvarupa wherein disease is yet to be manifested fully. The excited dosha having extended to other parts of the body become localized and it marks the beginning of specific diseases pertaining to those sthan/ structures. It is also known as the stage of disease augmentation. Sthana samshraya means taking shelter in a place.

In case of fever PGE2 is the ultimate mediator of the febrile response. PGE2 acts on neurons in the preoptic area (POA) through the prostaglandin E receptor 3 (EP3). EP3-expressing neurons in the POA innervate the dorsomedial hypothalamus (DMH), the rostral raphe pallidus nucleus in the medulla oblongata (rRPa), and the paraventricular nucleus (PVN) of the hypothalamus. Fever signals sent to the DMH and rRPa lead to stimulation of the sympathetic output system, which evokes non-shivering thermo-genesis to produce body heat and skin vasoconstriction to decrease heat loss from the body surface. It is presumed that the innervations from the POA to the PVN mediates the neuroendocrine effects of fever through the pathway involving pituitary gland and various endocrine organs.

In case of atherosclerosis sthansansraya takes place in myocardial vessel leads to angina/ myocardial ischaemia/ infarct. If it takes place in brain it leads to Cerebro

Vascular Event and if it takes place in peripheral vessel it leads to peripheral vessel disease.

If the inflammatory mediators attack the component of muscle it leads to myopathy whereas if intestine are involved it leads to Inflammatory Bowel Disease.

With respect of atherosclerosis when plasma LDL concentrations become elevated, the vessel wall eventually becomes lipid-engorged because it is unable to traffic the large amounts of endocytosed LDL-CE. In addition, lipoprotein entrapment by the extracellular matrix can lead to the progressive oxidation of LDL because of the action of lipoxygenases, reactive oxygen species, peroxynitrite, and/or myeloperoxidase found in oxidized LDL particles. A range of oxidized LDL species is thus generated, ultimately resulting in their delivery to vascular cells through several families of scavenger receptors. These "molecular Trojan horses" and "cellular saboteurs," once formed or deposited in the cell, can contribute to, and participate in, formation of macrophage- and smooth muscle-derived foam cells.

The accumulation of the WBCs is termed "fatty streaks" early on because of the appearance being similar to that of marbled steak. These accumulations contain living, active WBCs (producing inflammation) and remnants of dead cells, including cholesterol and triglycerides. The remnants eventually include calcium and other crystallized materials within the outermost and oldest plaque. The "fatty streaks" reduce the elasticity of the artery walls. However, they do not affect blood flow for decades, because the artery muscular wall enlarges at the locations of plaque. The wall stiffening may eventually increase pulse pressure; widened pulse pressure is one possible result of advanced disease within the major arteries.

Atherosclerosis is therefore a syndrome affecting arterial blood vessels due to a chronic inflammatory response of WBCs in the walls of arteries. This is promoted

by low-density lipoproteins (LDL, plasma proteins that carry cholesterol and triglycerides) without adequate removal of fats and cholesterol from the macrophages by functional high-density lipoproteins (HDL). It is commonly referred to as a "hardening" or furring of the arteries. It is caused by the formation of multiple atheromatous plaques within the arteries

Thus depending on srotovaigunya or khavaigunya or depending on organ/ system disease is caused.

In acute inflammation, after resultant movement of plasma into the tissue which lead to resultant stasis due to increase in the concentration of the cells within blood. Stasis allows leukocytes to marginate (move) along the endothelium, a process critical to their recruitment into the tissues.

**VYAKTI:** This stage may be stated to be that of manifestation of the fully developed disease- the resultant dosha dushya samurchana.

In case of fever the brain ultimately orchestrates heat effector mechanisms via the autonomic nervous system. It causes increased heat production by increased muscle tone, shivering and hormones like epinephrine (adrenaline) and also prevents heat loss by way of vasoconstriction.

In acute inflammation the increased collection of fluid into the tissue causes it to swell (edema). The main symptoms of the inflammatory response are as follows.

The tissues in the area are red and warm, as a result of the large amount of blood reaching the site.

The tissues in the area are swollen, again due to the increased amount of blood and proteins that are present.

The area is painful, due the expansion of tissues, causing mechanical pressure on nerve cells, and also due to the presence of pain mediators.



Specific patterns of acute and chronic inflammation are seen during particular situations that arise in the body, such as when inflammation occurs on an epithelial surface, or pyogenic bacteria are involved.

**Granulomatous inflammation:** Characterized by the formation of granulomas, they are the result of a limited but diverse number of diseases, which include among others tuberculosis, leprosy, sarcoidosis, and syphilis.

**Fibrinous inflammation:** Inflammation resulting in a large increase in vascular permeability allows fibrin to pass through the blood vessels. If an appropriate procoagulative stimulus is present, such as cancer cells, a fibrinous exudate is deposited. This is commonly seen in serous cavities, where the conversion of fibrinous exudate into a scar can occur between serous membranes, limiting their function. The deposit sometimes forms a pseudo-membrane sheet. During inflammation of the intestine (Pseudo-membranous colitis), pseudo-membranous tubes can be formed.

**Purulent inflammation:** Inflammation resulting in large amount of pus, which consists of neutrophils, dead cells, and fluid. Infection by pyogenic bacteria such as staphylococci is characteristic of this kind of inflammation. Large, localized collections of pus enclosed by surrounding tissues are called abscesses.

**Serous inflammation:** Characterized by the copious effusion of non-viscous serous fluid, commonly produced by mesothelial cells of serous membranes, but may be derived from blood plasma. Skin blisters exemplify this pattern of inflammation.

**Ulcerative inflammation:** Inflammation occurring near an epithelium can result in the necrotic loss of tissue from the surface, exposing lower layers. The subsequent excavation in the epithelium is known as an ulcer.

Atherosclerotic lesions, or atherosclerotic plaques, are separated into two broad categories: Stable and unstable (also called vulnerable). The pathobiology of atherosclerotic lesions is very complicated but generally, stable atherosclerotic



plaques, which tend to be asymptomatic, are rich in extracellular matrix and smooth muscle cells, while, unstable plaques are rich in macrophages and foam cells and the extracellular matrix separating the lesion from the arterial lumen (also known as the fibrous cap) is usually weak and prone to rupture. Ruptures of the fibrous cap expose thrombogenic material, such as collagen, to the circulation and eventually induce thrombus formation in the lumen. Upon formation, intraluminal thrombi can occlude arteries outright (e.g. coronary occlusion), but more often they detach, move into the circulation and eventually occluding smaller downstream branches causing thromboembolism. Apart from thromboembolism, chronically expanding atherosclerotic lesions can cause complete closure of the lumen. Chronically expanding lesions are often asymptomatic until lumen stenosis is so severe (usually over 80%) that blood supply to downstream tissue(s) is insufficient, resulting in ischemia.

**BHEDA:** It is the stage in which the disease may become sub-acute and chronic or incurable. Different types or variant of disease gets manifested.

In case of fever signs like increased blood pressure, neck stiffness, headache, giddiness, unconsciousness etc are seen in this phase.

In case of inflammation the outcome is manifested as:

**Fibrosis:** Large amounts of tissue destruction, or damage in tissues unable to regenerate, cannot be regenerated completely by the body. Fibrous scarring occurs in these areas of damage, forming a scar composed primarily of collagen. The scar will not contain any specialized structures, such as parenchymal cells, hence functional impairment may occur.

**Abscess Formation:** A cavity is formed containing pus, an opaque liquid containing dead white blood cells and bacteria with general debris from destroyed cells.

**Chronic inflammation:** In acute inflammation, if the injurious agent persists then chronic inflammation will ensue. This process marked by inflammation lasting

many days, months or even years, may lead to the formation of a chronic wound. Chronic inflammation is characterized by the dominating presence of macrophages in the injured tissue. These cells are powerful defensive agents of the body, but the toxins they release (including reactive oxygen species) are injurious to the organism's own tissues as well as invading agents. As a consequence, chronic inflammation is almost always accompanied by tissue destruction.

The importance of the scheme of kriyakala in early diagnosis and for adopting preventive and curative measures can be appreciated better by taking into consideration some of the recent trends in the modern medicine relating to the pathogenesis of disease.

Avoid hetu which are caused of dosha vridhi. Natural antioxidants (i.e.  $\beta$ -carotene, vitamin C, and vitamin E) have been used as a potential strategy to reduce damage caused by oxidized LDL in patients with or at high risk for CHD, but the majority of clinical trials have not shown reductions in CHD events with this approach. More clinically reliable markers of oxidative stress or the development of more effective antioxidant therapies might make this strategy more useful.

Rakta should always be given importance in all diseases. The mode of prasar should be assessed and managed at the same level.

Elevated values of circulating inflammatory markers such as CRP, serum amyloid A, IL-6, and IL-1 receptor antagonist commonly accompany ACS. Such elevations correlate with in-hospital and short-term adverse prognosis and may reflect not only a high prevalence of myocardial necrosis, ischemia-reperfusion damage, or severe coronary atherosclerosis but also a primary inflammatory instigator of coronary instability.

Non communicable disease are the main concerned in the 21st century, Metabolic Syndrome is among the main factor thus early diagnosis and prevention taken by changing the lifestyle can improve the health of society.

Study can be made by using specific antibiotics or specific medicine for each stage of evolution of disease. It will surely reduce the chances of drug resistancy and also control the vigorous use of drugs.

Study of shatkriyakala specifically sthanasansraya will help to understand kha vaigunyakar causes and help prevention of such hetus from causing the disease.

Conclusion: The utility of this shatkriyakala is to enable the treating physician to recognize the disturbances in its early formative stages and to enable to take necessary steps in time, to correct and eliminate the offending factors before they have caused sufficient damage.

8 may 2016

The 'upadrava' word is composed of two terms viz., 'upa' meaning afterwards or near and 'drava' (dravati-upaiti) means to appear. Thus the derivation of this word is an ailment which appears after or near due to main disease. Upadrava may be outsized or undersized. An ailment which appears after innovative disease as well as his roots are in pioneer disorder and involved with same is considered as aupsargik or upadrava. In visarpa, dōṣa are (bahu-upadravatayai) powerful and vitiation level is higher and generally associated with 'upadrava' as well as rōga sankar ,therefore , upadrava concept is described in visarpa cikitsā chapter.

Pramehapidaka occurs as a complication or upadrava of prameha. Chronic alcoholic liver cirrhosis may lead to portal hypertension and bleeding varices as a complication or upadrava. These both examples clarify the concept of upadrava.

Some times because of excessively aggravated dōṣa, several other disorders are manifested, in addition to the original disease. This condition of several diseases manifested concurrently, is called rōgasankara and is different from upadrava.

In sthūla various disorders occurs due to vitiated dōṣa like kustha,visarpa, bhagandara, jvara, atisār, meha, arsha, slipada, apache, kāmālā etc.This example helps to know the concept of rōgasankara.



Astanindita purusha includes atidīrgha (gigantism), atihrasva (short stature), atiloma (excessive body hair), alōmā (glabrous skin), atikṛṣṇa (hyperpigmentation), atigaura (hypopigmentation/ albinism), atisthūla (obesity), atikṛśa (lean and thin/cachexia).

1. Atidīrgha: Gigantism is over-secretion of growth hormone wherein there is marked increase in height and hypogonadism. Other features resemble acromegaly.
2. Atihrasva: Short stature (Dwarfism) may be caused by GH deficiency, hypothyroidism, Cushing's syndrome, precocious puberty, malnutrition, chronic illness, or genetic abnormalities that affect the epiphyseal growth plates (e.g., FGFR3 or SHOX mutations).
3. Atikṛṣṇa: Hyperpigmentation is one of the features of adrenal insufficiency. Hyperpigmentation may be striking or absent. Cortisol deficiency causes increased pituitary ACTH and MSH secretions which are responsible for the mucocutaneous accumulation of melanin.
4. Atigaura: Disorders of hypopigmentation are often classified as either diffuse or localized. The classic example of diffuse hypopigmentation is oculocutaneous albinism (OCA).
5. Atiloma: Hair can be categorized as either vellus (fine, soft, and not pigmented) or terminal (long, coarse, and pigmented). Excessive hair growth, it is known as Hirsutism in females, defined as excessive male-pattern hair growth, which affects approximately 10% of women. It usually represents a variation of normal hair growth, but rarely is it a harbinger of a serious underlying condition.
6. Aloma: Glabrous skin (hairless skin). Hypogonadism needs to be considered.



7. Atikṛśā: Lean and thin personality also known as cachexia. Hypocaloric states are associated with Growth Hormone resistance; abnormalities of Growth Hormone (GH) synthesis or action (e.g., pituitary failure, GHRH receptor defect, or GH receptor defect) reduce IGF-I (insulin like growth factor) levels leading to cachexia.

8. Atisthūla: Obesity results from relative excess calorie intake over caloric expenditure alongwith following endocrinal factors such as hypothalamic lesion which alter insulin levels and lipogenesis, independent of changes in food intake. Hypothyroidism, Cushing syndrome and hyperinsulinemia are the other endocrinological causes.

Caraka explained that on basis of sharir as adhikaran swaroop eight diseases are classified and among this eight atisthūla and atikṛśā are given utmost importance.

## **Endocrinology - AYURVED PERSPECTIVE**

Endocrine glands (endo- within) secrete their products (hormones) into the interstitial fluid surrounding the secretory cells rather than into ducts. From the interstitial fluid, hormones diffuse into blood capillaries and blood carries them to target cells throughout the body.

The endocrine glands include the pituitary, thyroid, parathyroid, adrenal, and pineal glands. In addition, several organs and tissues are not exclusively classified as endocrine glands but contain cells that secrete hormones. These include the hypothalamus, thymus, pancreas, ovaries, testes, kidneys, stomach, liver, small intestine, skin, heart, adipose tissue, and placenta. Taken together, all endocrine glands and hormone-secreting cells constitute the endocrine system.

The science related to the structure and function of the endocrine glands and the diagnosis and treatment of disorders of the endocrine system is endocrinology

Endocrinological disorders described today have their comparative counter parts in asthioniditiya adhyaya of Caraka Samhita. Further Prameha is the metabolic disorder mentioned in the text and as the management of endocrine disorders requires a broad understanding of intermediary metabolism, reproductive physiology, bone metabolism, and growth the sthula and sukshma pachana description mentioned in Grahani chikitsa adhyaya helps to understand the concept of endocrine system related disorders and their treatment.

The endocrine system as like nervous system controls body activities by releasing mediators, called hormones. The term hormone, derived from a Greek phrase meaning "to set in motion," aptly describes the dynamic actions of hormones as they elicit cellular responses and regulate physiologic processes through feedback mechanisms.

Hemadri defines chala guna as 'prerane chala' i.e. to set in motion. Chala guna is present in vata dosha therefore considering the nirukti of the word hormone it

resembles to one of the quality of vata. One can compare hormone to a vata dharmiya dravya.

**Hormones have the following effects on the body:**

Stimulation or inhibition of growth (vayu tantra yantra dhara/ pravartaka chestanam).

Wake-sleep cycle and other circadian rhythms (Santana gati vidhanam).

Mood Swings (niyanta pranetacha manasa/ harsha utsaho yoni).

Induction or suppression of apoptosis (programmed cell death), (aayusyo anuvritti pratyaya bhuta) (bhava abhavadakara).

Regulation of metabolism (samirano agne).

Preparation of the body for mating, fighting, fleeing, and other activity (pravartaka chestanam ucchavachanam).

Control of the reproductive cycle (udbhedanam cha udbhedanam)

Hunger cravings (samirano agne)

Sexual Arousal (apan karma)

A hormone may also regulate the production and release of other hormones/ (pranodana samana apana vyanat apananam).

Hormone signals control the internal environment of the body through homeostasis/ (aayusyo anuvritti pratyaya bhuta/ yantra tantra dhara)

Hormones are chemical messengers released from endocrine glands that coordinate the activities of many different cells. Coordination is of multiple organs

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and systems (srotas) from Central Nervous System to Excretory System. Srotas has been defined as channels in which parinama and abhivahan takes place. Three factors present in srotas viz: anupahat dhatushma, anupahat marut and anupahat srotas help to maintain sukha ayu, bala varna etc. The coordination between the srotas is brought about by nervous system and endocrine system. Both have the capacity to initiate and inhibit the action thus maintaining the coordination. Nervous system coordinates with help of nerve impulse whereas the endocrine coordinates with the help of hormones which are secreted within the interstitial fluid surrounding the secretory cells which through blood vessels reach the target organs where they carry-out the initiatory or inhibitory action.

Hormone release in the hypothalamus and pituitary is regulated by numerous stimuli and through feedback control by hormones produced by the target glands (thyroid, adrenal cortex and gonads). These integrated endocrine systems are called 'axes.' Caraka has explained integration (deham tantrayate samyak) with the help of vata and its five types. Although mulasthan of 5 types of vata have been explained at different sites in the body all are interrelated i.e. the reason why paraspara avaran has been mentioned.

A stressor is a chemical or biological agent, environmental condition, external stimulus or an event that causes stress to an organism. Stressors have physical, chemical and mental responses inside of the body. Physical stressors produce mechanical stresses on skin, bones, ligaments, tendons, muscles and nerves that cause tissue deformation and in extreme cases tissue failure. Chemical stresses also produce biomechanical responses associated with metabolism and tissue repair. Stressor stimulates the hypothalamus. Astangsangrahaakar has mentioned dhi, dhriti, smriti, mano bhodhan as karma of udan i.e. to analyze the situation, for eg. If snake is far away from the body there is no fight or flight situation whereas if it is next to the body there is sudden fight or flight condition. It means udan vayu helps mana to get avabodhan of the surrounding. It can be compared with analyzing the feedback signal received from various body organs and systems. The



situation is analyzed and signal is sent to hypothalamus where pran vayu takes the decision for inhibitory or initiative action to be taken this is understood by the dharan karma of budhi and chitta i.e. mana. Hypothalamus secretes the corticotropin release factor (crf) which stimulates the pituitary gland to release adrenocorticotrophic hormone ("ACTH"). Udan vayu by its prayatna and urja karma helps in the secretion. The release factor "ACTH" is taken to adrenal cortex with the help of vyan vayu. Vyan vayu is said to be shigrakari or fast acting i.e. within fraction of seconds ACTH reaches the adrenal cortex. Adrenal Cortex secretes various stress hormones which is stimulated by saman vayu. Mulasthan of saman vayu is in sweda, dosha, ambhuvaha srotas, it signals for the saman anayati karma i.e. to maintain homeostasis thereby releasing the hormones in blood stream. The stress hormone released in blood is again taken by vyan vayu to various organs like heart, intestine etc. to cause the flight-or-fight response. After the response the apan vayu comes into action to excrete the hormones and neutralized the effect.

Between this flow there is an alternate path that can be taken after the stressor is transferred to the hypothalamus (udan and pran karma), which leads to the sympathetic nervous system (vyan vayu). After which, the adrenal medulla secretes epinephrine (saman vayu) in blood and with the help of vyan vayu spreads throughout the body to cause the flight or fight response.

13 Feb 2016

### **My approach to hormones in ayurveda perspective ;**

Hormones are chemical messengers released from endocrine glands that coordinate the activities of many different cells. Coordination is of multiple organs and systems (srotas) from Central Nervous System to Excretory System. Srotas has been defined as channels in which parinaman and abhivahan takes place. Three factors present in srotas viz: anupahat dhatushma, anupahat marut and anupahat srotas help to maintain sukha ayu, bala varna etc. The coordination

between the srotas is brought about by nervous system and endocrine system. Both have the capacity to initiate and inhibit the action thus maintaining the coordination. Nervous system coordinates with help of nerve impulse whereas the endocrine coordinates with the help of hormones which are secreted within the interstitial fluid surrounding the secretory cells which through blood vessels reach the target organs where they carry-out the initiatory or inhibitory action.

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{My approach : integrated panchātmā vāta :

Hormones are chemical messengers released from endocrine glands that coordinate the activities of many different cells. Coordination is of multiple organs and systems (srotas) from Central Nervous System to Excretory System. Srotas has been defined as channels in which parinamana and abhivahan takes place. Three factors present in srotas viz: anupahat dhatushma, anupahat marut and anupahat srotas help to maintain sukha ayu, bala varna etc. The coordination between the srotas is brought about by nervous system and endocrine system. Both have the capacity to initiate and inhibit the action thus maintaining the coordination. Nervous system coordinates with help of nerve impulse whereas the endocrine coordinates with the help of hormones which are secreted within the interstitial fluid surrounding the secretory cells which through blood vessels reach the target organs where they carryout the initiatory or inhibitory action.



Hormone release in the hypothalamus and pituitary is regulated by numerous stimuli and through feedback control by hormones produced by the target glands (thyroid, adrenal cortex and gonads). These integrated endocrine systems are called axes.' Caraka has explained integration (deham tantrayate samyak) with the help of vata and it's five types. Although mula sthan of 5 types of vata have been explained at different sites in the body all are interrelated i.e. the reason why paraspara avaran has been mentioned.

A stressor is a chemical or biological agent, environmental condition, external stimulus or an event that causes stress to an organism. Stressors have physical, chemical and mental responses inside of the body. Physical stressors produce mechanical stresses on skin, bones, ligaments, tendons, muscles and nerves that cause tissue deformation and in extreme cases tissue failure. Chemical stresses also produce biomechanical responses associated with metabolism and tissue repair. Stressor stimulates the hypothalamus. Astangsangrahar has mention dhi, dhriti, smriti, mano bhodhan as karma of udana i.e. to analyze the situation, for eg. If snake is far away from the body there is no fight or flight situation whereas if it is next to the body there is sudden fight or flight condition. It means udan vayu helps mana to get avabodhan of the surrounding. It can be compared with analyzing the feedback signal received from various body organs and systems. The situation is analyzed and signal is sent to hypothalamus where prana vayu takes the decision for inhibitory or initiative action to be taken this is understood by the dharan karma of budhi and chitta i.e. mana. Hypothalamus secretes the crf (corticotropin release factor) which stimulates the pituitary gland to release "ACTH" (adrenocorticotrophic hormone). Udan vayu by its prayatna and urja karma helps in the secretion. The release factor "ACTH" is taken to adrenal cortex with the help of vyan vayu. Vyan vayu is said to be shigrakari or fast acting. i.e. within fraction of seconds ACTH reaches the adrenal cortex. Adrenal Cortex secretes various stress hormones which is stimulated by saman vayu. Saman mulasthan is in sweda, dosha, ambhu vaha srotas, it signals for the saman anayati karma i.e. to maintain homeostasis thereby releasing the hormones in blood stream. The stress



hormone released in blood is again taken by vyan vayu to various organs like heart, intestine etc. to cause the flight-or-fight response. After the response the apana vayu comes into action to excrete the hormones and neutralized the effect.

Between this flow there is an alternate path that can be taken after the stressor is transferred to the hypothalamus (udana and prana karma), which leads to the sympathetic nervous system (vyan vayu). After which, the adrenal medulla secretes epinephrine (saman vayu) in blood and with the help of vyan vayu spreads throughout the body to cause the flight or fight response}.

One science , one meaning , through two ways of interpretation :

The endocrine system, like nervous system, controls body activities by releasing mediators, called hormones. The term hormone, derived from a Greek phrase meaning “to set in motion,” aptly describes the dynamic actions of hormones as they elicit cellular responses and regulate physiologic processes through feedback mechanisms.

Hemadri defines ‘Chala guna’ as ‘prerane chala’ i.e. to set in motion. ‘Chala guna’ is present in Vata dosha , therefore considering the ‘nirukti’ of the word hormone, it resembles to one of the quality of vata. One can compare hormone to a Vata dharmiya dravya ( a substance endowed by Vata) ..}}

29 Jan 2015

### Thyroid diseases

In thyroid diseases, consider samaana and udaana.. its endocrine disorders.. thyroid hormones induce calorogenesis through promoting basal metabolism.. the role of samaana is like thyroid hormones to promote agni work, to help in sweating and the role of udaana is prayatna, bala and urjaadi... urjaa and in turn bala comes after calorogenesis.. in hyperthyroidism, hypermetabolic state is present, means the action of samaana is increased, due to increased catabolism bala n urjaa are decreased so it can be correlated with samaana avrita udaana.. kanchanar shigru bakuchi manjishtha shatavari ashwagandha svarna tamra like drugs are helpful.. in hypothyroidism, hypometabolic state is present, it means the action of samaana is decreased.. so it can be correlated with udaana avrita samaana.. chatushparni punarnava chitraka trikatu gokshuru mandoora svarnamakshika like drugs can be used.. increased appetite with weight loss excessive sweating hyperdefecation heat intolerance glucose intolerance in hyperthyroidism due to stimulated samaana.. tachycardia systolic hypertension, IHD, Heart failure, myopathy, weakness due to aavrita udaana... in hypothyroidism decreased appetite with weight gain, decreased sweating cold intolerance hyperlipidaemia constipation ileus due to aavrita samaana ie decreased functioning of samaana vaata. Bradycardia diastolic hypertension myxedema are due to vaikrita udaana... Hormonal changes influence cellular metabolism so latter multiple dearrangement occurs as consequences of primary changes.. its my hypothesis.. jatamansi will help in hyperthyroidism if insomnia is present... for hypersomnolence as in hypothyroidism, chitraka trikatu vacharasona like drugs may be helpful...Kanchanara guggula arogyavardhini vati chandraprabha vati punarnavadi mandoora chitrakadi vati lekhaneya kashaya chatushparni prabhakar vati chitrakadi vati shigru patra kwath maha manjisthaadi kwath are found effective even without modern drugs.. few vaidya recommend

vaman virechan raktamokshana too...In presence of tacharrythmia ( in hyperthyroudism ) prabhakar vati with arjun shatavari and brahmi are useful..

## Cushing syndrome

Cushing syndrome is one among the cause for atisthūla. Cushing's syndrome, also known as hypercortisolism, Itsenko-Cushing syndrome, and hyperadrenocorticism, is a collection of signs and symptoms due to prolonged exposure to cortisol.

### Aetiological factors

Iatrogenic cause: taking glucocorticoids prescribed by a health care practitioner to treat other diseases. This can be an effect of corticosteroid treatment of a variety of disorders such as asthma and rheumatoid arthritis, or in immunosuppressant after an organ transplant. Cushing's syndrome in childhood usually results from use of glucocorticoid medication.

Endogenous cause: Pituitary corticotrope adenomas account for 70% of patients with endogenous causes of Cushing's syndrome.

Ectopic tumor ACTH production: cortisol-producing adrenal adenomas, adrenal carcinoma, and adrenal hyperplasia account for the other causes; rarely, ectopic tumor CRH production is encountered. Tumors outside the normal pituitary-adrenal system can produce ACTH (occasionally with CRH) that affects the adrenal glands.

Pseudo-Cushing's syndrome: Elevated levels of total cortisol can also be due to estrogen found in oral contraceptive pills that contain a mixture of estrogen and progesterone, leading to Pseudo-Cushing's syndrome.

### Pathophysiology of Cushing syndrome

Paraventricular nucleus (PVN) of the hypothalamus (pran vayu analyze the situation)

(udan vayu by its prayatna and urja karma alongwith pran vayu helps in release of hormone)

Corticotropin-releasing hormone (CRH)



Stimulates the pituitary gland to release  
Adrenocorticotropin (ACTH)  
Travels via the blood (vyano rasa dhaturhi vikshepa uchit karmanaha)  
Adrenal gland  
ACTH stimulates the release (Samana)  
Cortisol      Elevated levels of cortisol      if able to analyze  
by udan vayu  
Exert Negative  
feedback  
If unable to analyze by udan vayu  
CRH in the hypothalamus

Hypercortisolism      ACTH released from the  
anterior pituitary gland.

Cushing Syndrome  
Normal Cortisol level  
Apan excretes and neutralizes  
the effect of Cortisol in blood

### **Symptoms of Cushing's syndrome**

Rapid weight gain: Meda eva upachiyate na tatha itare dhatawa. Chala sphika sthana udara lambhana (central obesity) are the features of sthaulya. Dalhan says meda janan (lipogenesis) occurs either due to aahar, adrishta vashad medasa avrita marga tvatcha i.e. due to then unknown reason wherein meda causes avaran of vata dosha leading to adipose deposition, particularly on the trunk and face with sparing of the limbs.

Thin and fragile skin: As upachaya of only meda dhatu takes place the mansa, rakta get depleted thereby hampering the lepana, and jeevan karma of mansa, and rakta respectively. This reduces the tensile strength of skin and makes it thin and fragile.

Purple or red striae: The weight gain in Cushing's syndrome stretches the skin, which is thin and due to weakening and rupture of collagenous fibers (lepan karma of mansa is hampered) in the dermis.

Easy bruising and ecchymosis (tiryaka vata pradhan raktapitta): Dilation of capillaries and thinning of the skin and mucous membranes leads to easy bleeding.

Proximal muscle weakness (hips, shoulders) is observed due to excessive protein catabolism. Caraka mentions that daurbalya is due to asamatva dhatunam i.e. anabolism of meda continues whereas catabolism of anya dhatu like mansa, asthi etc dhatu is observed.

Osteoporosis is also an issue in Cushing's syndrome since osteoblast activity is inhibited. Mulasthan of asthivaha srotas is meda and jangha. Vikruta Meda causes reduced poshakansa for asthi dhatu causing asthi kshaya i.e. protein matrix of bone is loss and there is increased of calcium excretion. Further asthi and vata has ashrayashrayi sambandha therefore when asthi kshaya takes place vata vridhi occurs leading to asthi sandhi shool. Cushing's syndrome may cause sore and aching joints, particularly in the hip, shoulders, and lower back. The fractures heal badly and are accompanied by excessive callus formation.

Hirsutism (facial male-pattern hair growth), baldness and/or extremely dry and brittle hair are observed due to excess of androgens. Androgen excess in women leads to increased hair growth in most androgen sensitive sites except in the scalp region, where hair loss occurs because androgens cause scalp hairs to spend less time in the anagen phase.

As discuss above since asthiposhakansa are less in quantity asthi kshaya takes place (depleting the concentration of calcium in the bone causing hairfall) and leading to increase of asthimala bhag (kesha and smashru vridhi). Role of rasa dhatu also needs to be studied as sukumar loma are rasa sara lakshana and Sushrut has mentioned rasa as cause for sthauilya and karshya.

Decreased fertility in men: Caraka mentions shukra abahutvat and medasa avritta marga to be cause for kricha vyavayata. Sushruta says due to sthauilya kapha and meda causes obstruction (nirudha) of shukra marga leading to alpa vyavaya (loss of libido) and since other than meda dhatu apyayana of other dhatu does not takes place leading to shukra kshaya leading to decreased fertility. Androgen is the cause for decreased fertility in male. Androgens are hormones (vata dharmi) and if one vata prakar is vitiated it can also vitiate the gati of other vayu prakar. In this case it is avritta apan.

Menstrual disorders such as amenorrhea/ oligomenorrhoea in women due to excess androgen: Due to rasa dushti and rakta and shukra alpata leads to alpa raja srava. Avritta vata as discuss above may also be the cause. Studies have also shown that the resultant amenorrhea is due to hypercortisolism, which feeds back onto the hypothalamus resulting in decreased levels of GnRH release (tantra yantra dhara karma of vata is loss and also balance functioning of panchatma vayu is disturbed)

Immune suppression: Bala depends on dhatu (dharanat dhatawa) as later on in the disease there is apachaya of all the dhatu leading to alpa bala.

Moodiness, irritability, or depression is observed: Niyanta praneta cha manasaha is karma of vata as vata gati is vitiated the prakrita karma of mana is disturbed. It also affects the memory and attention dysfunction. Rate of suicide is increasing due to such psychological changes.

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Other symptoms include excess sweating (swedadhikya Su. Su. 15/32) and sleep disturbances.

#### **Treatment:**

Nidan parivarjanam: a) drugs like glucocorticoids should be stopped.

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Medicines and diet regimen should be such that it will do yapana (maintaining) of agni but at same time it will cause emaciation. In sthaulya, agni sandukshan (alleviated power of agni) is present which is cause for aahar shoshan leading to increased appetite. Guru aahar will prevent fast digestion of aahar and due to atarpan quality will not be doing bruhan.



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Low-glycemic carbohydrates can also help lower cortisol levels naturally. Lower glycemic choices include brown rice, wheat bread and pasta, sweet potatoes, beans, and fruits and vegetables. Eat carbohydrates immediately following exercise to blunt the surge of cortisol that intense activity causes. High-quality sources of protein can help to decrease cortisol production. Whey protein, eggs and lean animal meats contain amino acids that are essential to survival. Other useful protein supplements include soy, rice, pea, hemp and vegetable proteins. Omega-3 fats from fish and flax seed oil can help to control stress. Fruits and vegetable contain many healthy phytonutrients and vitamins that can inhibit cortisol production. For example, vitamin C from citrus fruits and greens has been shown to be very effective at decreasing cortisol production

### **Medicines:**

Musta (Cyperus rotundas)

- Stress-Reducing Effects: Herbal mixture that included CR seems to help reduce stress-related physiological and psychological symptoms.

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23 March 2016

Musta can be used in Cushing syndrome ;

- Stress-Reducing Effects: Physiological effects of herbal mixture with *Cyperus rotundus* L. on blood pressure, norepinephrine, cortisol and psychological variables in healthy medical students under examination: Herbal mixture that included CR seems to help reduce stress-related physiological and psychological symptoms.

Anti-Obesity Effect: Study of *C. rotundus* tubers hexane extract for 60 days induced a significant reduction in weight gain without affecting food consumption or inducing toxicity. In vitro, the extract was able to stimulate lipolysis. The effect in weight gain may be partially mediated through activation of  $\beta$ 3-AR. Results suggest CR tuber extract has a potential as a herbal supplement for controlling body weight. Administration of *Cyperus rotundus* tubers extract prevents weight gain in obese Zucker rats / Bernard Lemaure et al.

Decreasing Hair Growth / Role in Androgenic Hair / Essential Oil: Study evaluated the efficacy and safety of Egyptian *C. rotundus* essential oil in decreasing androgenic hair (hirsutism and axillary hair) in 91 female patients. Results showed topical application of essential oil is an effective method in treating moderate degrees of hirsutism and axillary hair., without affecting serum testosterone. • Lipid Lowering / Rhizomes: Study of alcoholic extract of rhizomes of *Cyperus rotundus* demonstrated statistically significant reduction of serum lipid profile.



14 July 2014

Acharya charak mentions; pramehi tathaa atasee sarshap taila yuktam (ch.chi.6/20), if pramehi is contraindicated for sanshodhanam...

Sushrut Ch. 11/6 (prameha chikitsa); Tikta kashaayaabhyaam cha shaakaganaabhyaam nikumbha ingudee sarshapa atasee taila sidhaabhyaam.

Both acharya indicate the use of atasee/alasee / flaxseed in prameha as pathya.. prameha is risk factor for atherosclerosis, IHD and Dyslipidaemia. These references favor modern observations..

11 July 2014

**Diabetes and chronic liver disease (CLD)** are common long-term conditions in the developed and developing world. The 2 conditions often coexist, and there is evidence to suggest that diabetes can have a significant adverse effect on patients with CLD, leading to increased complications and premature mortality. While diabetes, nonalcoholic fatty liver disease, and nonalcoholic steatohepatitis (NASH) appear to have common origins related to obesity and insulin resistance, diabetes is also common among patients with alcoholic and viral CLD. In patients with NASH, improvement in metabolic indices appears to reduce the progression of CLD. It is not clear whether improving glycemic control in other forms of CLD leads to improved outcomes. Managing diabetes in patients with CLD can be challenging because many antihyperglycemic therapies are contraindicated or must be used with care. Metformin and pioglitazone may be useful in patients with NASH, but sulfonylureas and insulin must be used with caution, as hypoglycemia may be a problem. Insulin doses frequently need to be reduced in patients with CLD. Newer glycemic agents have not been widely used in patients with CLD, but bariatric surgery may lead to significant improvement in liver indices in patients with NASH. Management of patients with diabetes and CLD may be enhanced by using a multidisciplinary approach. The drugs of tikta rasa

are useful in both coexisted diseases ; kiraatatikta, patola , amrita , vaasaa , arjuna , saptaparni , bhoomyaamalaki , gudamaara , vijayasaar etc...

Sarv ev prameha yasmad deham madhuraikritya jayante tasmad madhumeha iti uchyante.. chakrapani on ch.su.17/3-7.. Diabetes Mellitus comprises a group of common metabolic disorders that share the phenotype of hyperglycemia....

22 Sept 2012

Chakrapani on ch.chi.6/57 ;

kulaja iti  
pitripitaamahaadikaranodbhutaah ..... .santaanaanubandhitvopadarsha naartham. uktam hi-pramehoanusanginaam (ch,su.25/40) iti.. first degree relatives have a higher risk of developing T1D than unrelated individuals from general population (Dorman and Bunker 2000).family studies have revealed that first degree relatives of individuals with T2D are about 3 times more likely to develop the disease than individuals without a positive family history of disease (Flores et al. 2003)

21 Dec 2014

Kleda is intermediary products /end products of metabolism which tend to accumulate in body.. kleda is produced either due to decreased anabolism or increased catabolism or both.. e.g. DM , CKD, Cushing's syndrome, Hypothyroidism, metabolic acidosis , Hyperkalaemic acidosis , lethogenesis, CCF, etc..

Kleda is components of physiological actions.. kleda is responsible to maintain osmolality of plasma for osmosis.. Hyperketonaemia in presence of deficiency of insulin leads to kleda production/ketoacidosis, however in normal person insulin utilizes ketones as fuel.. Long term hyperglycaemia in DM leads to glycosylation

and glycation of intracellular matrix ie kledaanvita meda maansaadi.. Ureamia is kleda induced multiorgans disease with failure of renal function.. being moola of medavaha srotas kidney has crucial role to convert kleda into mootra as per metabolic status ; less urine in dehydration and more urine in polydipsia..

17 Sept 2012

Sharir kledam punardushayan mutratven parinamayati, mutravahanam cha srotasam vankshan bastiprabhavanam medahkledophitani gurooni mukhanyasadya pratirudhyate;ch.ni 4/8 chakrapani; aasadya pratirudhyat iti gatva avatishthate. Hyperglycaemia(>180mg/dl)- Glucosuria-osmotic diuresis-polyuria & nocturia

Kaalameha , is pittaja prameha , in which black urine is excreted in large amount , can be correlated with Alkaptonuria ( black urine disease , black bone disease ) is a rare inherited genetic disorder of phenylalanine and tyrosine metabolism. Due to defect in enzyme homogentisic acid dioxygenase , homogentisic acid and its oxide ,called alkapton ,accumulate in the blood and are excreted in urine in large amount. Urine may turn black if collected and left exposed to open air. Excessive homogentisic acid causes damage to cartilage ( ochronosis- osteoarthritis) and heart valves as well as precipitating as kidney stone.. t/t nitrofurantoin , a suppressor of enzyme .. ayurved perspective ; pittaja prameha -apatarpana-vaata prakopasandhigatavaata. Due to vitiated pitta kidney stone and heart valves damage.. t/t.. as per pittaja prameha in ch.chi.6/30-32; ushira , lodhra, arjun ,patola, nimba, aamalaki , amritaa ,abhayaa, musta, bilva , asana ,kamala ,padmyakaashtha etc..

24 April 2013

Mutra shyav varna. Ch.ind.. Blackish urine...as in Alkaptonuria , disease of tyrosine catabolism.

10 April 2015

### **Acute pancreatitis**

Gall stones alcohol etc - pitta prakopa in term of ushna tikshna vridhhi - paaka karma vridhhi in agni sthaana \*(pittaja gulma )- agnisaada - (pittavrita samaana). shoola jvara chhardi due to pitta prakopa , indigestion and malabsorption due to aavrita samaana . Due to pitta vridhhi vaata marga avarodha leading to rakta pitta sanchiti in udara ; jalodara.. sanga pradhaan dushti so eka sthaana vridhhi (jalodar) and itar (other ) sthaana kshaya - circulatory failure.. excessive pitta prakopa - rakta dushti - paaka- septicaemia..

Pitta prakopa and vaata gati hanana result in gulma and jalodara , aavrita samaana leading to agnisaada.. apatarpana is severely present.. managament ; laghu santarpana , deepaniya yavaagu , shrita yoosha , shadanga paaniya , pitta shamana , agni vardhana , due to chhardi amalaki mashi with pravala or shankh bhasma and mayurapuchchha bhasma muhurmuuh , especialy before intake of fluid, food and drugs

Already apatarpana so sanshodhana chikitsa is contraindicated , tiktaksheera ghrita basti /yaapana basti can be considered to work on pitta & vaata. There is exocrine insufficiency - less pancreatic enzyme secretion - indigestion & malabsorption - diarrhea and steatorrhea ( in acute phase severe abdominal pain vomiting and fever ) - agni vardhan chikitsa will decrease the load on pancrease , hence recovery from inflammation will be fast.. Electrolytes and water maintainance is crucial with nutritional support ; shrita yoosha / deepaniya yavaagu are best choice ..

Swarna makshika bh 10 grm+

Pravalapisti 10 grm+

Guduchi satwa 10 grm+



Amlaki churna 30 grms

1 gm twice daily.

On 2-3 days pain intensity gradually drops.lashunaadi vati like drugs are effective.. aamalaki mashi combination is effective in chhardi.In acute pancreatitis (pitta prakopa ) due to inflammatory degeneration of exocrine cells , enzyme secretion is very less \*(aavrita samaana), so indigestion and malabsorption (agnisaada) occurs

Agniboosting drugs which provide enzymes to help in digestion so load on pancrease is decreased providing time for healing.. Lashuna ksheerapaaka is mentioned in gulma with indication in antarvidradhi...For samaana boosting chitraka lashuna like drugs are found effective , its not hypothesis , its observation.. Pulling of fluid in peritoneum indicates the role of vaata.These drugs are also helpfull in removing obstructive pathology

. Jalodar is another consequence present in pancreatitis excludes paittik shoola ..ERCP with stent placement is also indicated for pancreatic ductul disruptions that occur as part of the inflammatory process and result in peripancreatic fluid collections...In addition to nutritional support , enteral feeding helps to maintain integrity of the intestinal tract during severe acute pancreatitis..

The maintainance of intestinal integrity is possible with drugs acting on agni or samaana vaayu..

Enteral feeding with a nasojejunal tube has been demonstrated have fewer infectious complications than with TPN and is preffered method of nutritional support.. Therefore i prefer the use of shadangapaaniya , shrita yoosha and laghu santarpana through nasojejunal tube in place of IV fluids and colloid to maintain normal intravascular volume... Analgesics for pain ; shankha vati /chitrakaadi vati /lashunaadi vati/ agnitundi vati..There is future prospectus in ayurveda to treat

such diseases which can cause multiple organ failure ; need to select appropriate treatment modalities.. thanks for discussion Gulma chikitsa in charak is best reference to understand the diseases related to GIT and their line of treatment ..Later in charak chikitsa 26 in reference to hridroga acharya charak mentions various shoola for differential diagnosis purpose... acharya madhav referred such shoola dominating diseases in details.. As parinaama shoola annadrava shoolaadi..

In sushruta uttaratantra after gulma hridroga chikitsa is classified and at the end of gulma description , acharya sushruta mentions hrichchhula , the purpose is differential diagnosis. Diagnosis is crucial to decide perfect line of treatment...

रक्तहरणार्थं रूपमाह रक्तस्य मार्गं मारुतो निहन्ति शाखासन्धिषु -  
, ततः च वायुना रक्तनिरोधात् , निविश्येति शाखासन्धिषु स्थित्वा  
वातरक्तं , अन्योन्यं आवर्येति रक्तेन् वातं वातेन् च रक्तं आवार्य  
अत एवात्र वातशोणिते असृक् विमुञ्चेत् . वेदनाभिः हरेदसूनिति योज्यम्  
.37-35/29 .चि .च - आचार्य चक्रपाणि . इति भावः

Physiologically, blood communicates at almost every zone of human body either directly or by indirect influences. The role and importance of Rakta (blood tissue) in the genesis, manifestation and progress of various clinical conditions with respect to Vataraktha is evident. Siravyadha is one such radical treatment especially concerned with Dustarakta nirharana (the macroscopic removal of 'morbid blood' from unwanted contexts or .(situations

Patho-physiological studies suggest that in case of a considerable blood loss (> 100 ml), the immediate haemo-dilution stimulates / triggers a host of beneficial physiological mechanisms making the body alert and adaptive to take care of various systemic challenges present. Back up support in the form of immunologic, inflammatory and trigger factors intended for specific purposes is recruited in pathological tissues for the management of the damage. After considerable amount of bloodletting, Psycho-Neuro-Endocrinal mechanisms mediated by Hypothalamus, Pituitary, and Adrenocortical axis are triggered. Brain responds with commanding actions through efferent signals to vessel or vascular system.

In Vataraktha which is a metabolic disorder of impairment of purine metabolism, serum uric acid level is high and also there is inadequate excretion i.e. nothing but hyperuricemia. Bloodletting in dorsal venous arch causes reduction in uric

acid. There may be release of angiotensin hormone which has got renal and adreno-cortical stimulatory effect providing excretion of uric acid by kidneys. Erythropoietin may be activated, which in turn successfully addresses most of the circulatory / vascular related healing drifts. The dusta raktha which is shaakhashrita in acute stages are expelled readily by Siravyadha as it is the nearest route for dosha nirharana. Shonitha Kleda is one of the Pittaja Nanatmaja vikara and here Kleda refers to multiple intermediate metabolites, particles etc, embedded in Rakta hence Raktamokshana by Siravyadha is an attempt to reduce the excessive kleda in Shonitha.

Even more “Raktam hi amlatam hi tatha cha ruk” means amlata in rakta (inflammatory markers, intermediate metabolites, acidic components like uric acid) is responsible for Ruk (all types of pain).

The complex activities which render pathological sequences leads to permanent and hallmark pathological features like Gouty arthritis. No drug or regimen is effective to dislodge these factors easily and effectively. The new ‘regional balance’ of morbidities in a disease will transiently get disturbed on to a positive plane by Siravyadhana followed by re-launching of healing mechanisms. In general, various probable mechanisms are going to change in body by bloodletting, such as local blood supply is improved, local metabolism is improved, local drainage system is improved, fresh RBCs are produced which are active. Hepatocellular activity in particular enzyme system is improved which results in correction of purine metabolism hence regulation of Uric acid generation. Release of hormones, sympathetic nerve function etc, are triggered which directly stimulates bone marrow and immune related T-Lymphocytes.

26 May 2016

लवणो रसः पित्तं कोपयति ; 3/42/26.सू.च , तर्षयति , रक्तं वर्धयति ,



Ati lavana sevan (<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2871322/>) Thirst is highly sensitive to increases in plasma sodium concentration and osmolality, requiring only a 2%–3% increase to induce feelings of thirst. A larger change in plasma volume (10%) is required to induce thirst if there is no concomitant change in plasma sodium concentration. If plain water is used to replenish body water, plasma volume is preferentially restored over the interstitial and intracellular fluid space, suppressing plasma sodium concentration and removing the dipsogenic drive long before total body fluid has been restored. During or after dehydrating exercise, sodium ingestion helps to maintain and restore plasma volume and osmolality by continuing thirst sensation (thus drinking) and also by increasing body fluid retention. A high sodium meal or intravascular hypertonic saline infusion may cause transient osmotically mediated blood pressure increases, but in healthy people, acute sodium ingestion does not cause sustained hypertension. The purpose of this review is to provide evidence that acute increase in sodium are an intrinsic part of the thirst response during and after exercise, and that blood pressure increases associated with hypertonicity appear to be short lived. To ensure the precise regulation of volume and osmolality of body fluids, a number of integrated neural and hormonal systems have evolved to control thirst and sodium appetite. These systems respond to stimuli that arise from a deficit of fluid in both the intracellular and extracellular fluid compartments or to systemic hypertonicity (in humans, plasma osmolality is primarily a function of plasma sodium concentration or “tonicity” of the blood). Osmotic stimuli are sensed by osmo-Na<sup>+</sup> receptors, which are nerves that are responsible for detecting the concentration of the interstitial fluid. Plasma osmolality elevation is the most potent stimulus of thirst, with only a 2%–3% change in osmolality required to induce thirst in humans.

29 Aug 2014

**Raktapitta** ; Tairhetubhi ; Amlo raso pittam abhivardhayati , raktam dooshayati . Lavano rasah pittam kopayati, raktam vardhayati. Katuko raso shonita sanghaatam bhinatti,pittam kopayati (ch.su26)- samutklishtam pittam -> raktam prapadyate-> pittasya vardhanam-> raktam pradooshayat -> tasya(pittasya ) ushmanaa svidyamaana maansaadi dhatubhih achyutena dravaroopena dhaatunaa yuktam Sapittam (Vikaara vighaata abhaava) -> bhooyo atitaraam vridhim gachchhati iti yojanaa ( chakrapani on ch.chi4/7-8)..treatment is concerned with tikta madhur kashaaya and sheeta drugs... like vaasaa , chandaana, ushira, dhaataki, prishnaparni, kiraatatikta, mustaa,lodhra , padmyakaashtha, draakshaa, madhuka, priyangu, patol, kamala,shataavari, shankha,pravaala, gairika , muktaa ,suvarna, aamalaki etc..

**Hemophilia** ; is an X-linked recessive hemorrhagic disease due to mutation in factor 8 (F8) gene (Hemophilia A) or factor 9 gene (Hemophilia B). These genes come from mother ( Rakta is maatrija bhaava) to their children , however male child is clinically affected and female child becomes asymptomatic carrier.. clinically , Hemophilia A and Hemophilua B are indistinguishable..The disease is characterized by bleeding into joints, muscles , soft tissues after minor trauma or even spontaneously. Prolonged aPTT assays , normal bleeding time , normal platelet counts. Diagnosis is made after specific determination of F8 or F9 clotting activity. Without treatment , severe hemophilia has a limited life expectancy(now yaapya, earlier pratyaaakheya before discovery of these factors). Recombinant F8 & F9 proteins are indicated as factor replacenet therapy for prophylactic and curative purpose.. In ayurveda tiryaka raktapitta , pratyaaakheya.. so life saving factor replacement therapy with raktapittaghna drugs **can be recommended..**

26 Nov 2014

**Clot formation and clotlysis- Ayurveda perspective ;**

Rakta sraava and kaphaanubandha ; after rakta sraava kapha comes in contact with bleeding site to ceal bleeding..kapha is prithvi and aapa mahaabhootabhuyishtha , so due to parthivaansha of kapha sanghaat develops at site of bleeding and aapaansha of kapha leads to bandhanam between parthivaansha and bleeding site , which in turn adheres parthivaansha at that of site , thereafter it results in sthairyam (immobility) to ceal bleeding.. Rakta sraava+ kapha parthivaansha sanghaata( kaathinya) aapaansha bandhanam ( paraspara yojanaa ) parthivaansha sthairyam ( avichaalyam ) clot/thrombus formation cealing of bleeding... the action of katu rasaatmaka drugs is \* shonita sanghaatam bhinnati\*, therefore katu rasaatmaka drugs are found effective in dissolution of clot and in fibrinolysis ( anticoagulant and fibrinolytic action )..

25 April 2016

## वातशोणित

Vātaśonita is the disease produced by vitiated vāta dośa and rakta which impede the gati of each other causing the disease. This disease is also renamed as ādhyaroga i.e. mainly affluent people are affected by the disease as said by Chakrapāṇi.<sup>1</sup> Very detailed aetiological factors had been mentioned in Caraka Samhita which were in practice during that very time and now has become either out dated or localized to very remote places in India, like pinyaka (in ancient time, the residue of oil seeds were eaten after oil had been extracted), madya like arnāl, sauvir, sukta, surā, āsava like alcoholic beverages which were in use which have been replaced by refined alcoholic drinks like beer, wine etc. Again during old time horse, camel and yāna (carts) were used for travelling which may be co related with travelling means of modern era. The disease is predominant among the people who in general are not habitual to physical activity rather they have more sedentary lifestyle. People of pitta prakriti (pitta constitution) are prone to develop vātaśonita, sukumāratwa (delicate personality) is the characteristic feature of pitta prakriti.

Each of the three dośa, which are physiological entity can be subdivided into three types depending on the mode of action which each of the three execute in the normal and pathological stages, a) normal physiological activities of the body are dhātu dośa (normal dośa), b) abnormal physiological activities of dośa in the body- (vitiating dośa) and c) by products of abnormal physiology will be the mala dośa (waste product) only which have anatomical entity and are removable from the body. All normal micro and macro movemental activities in the body are dhātu vāta, all the activities responsible for heat production and regulation are dhātu pitta and all the synthesis or productive activities of the body are dhātu kapha. Dośa (normal physiological activities) are very essential for the body so in that context they are called as dhātu, but when they are prone to vitiation they



are called as dośa. All abnormal movemental (macro and micro) activities in the body are caused by vitiated vāta dośa, abnormality in heat production and regulation in the body are caused by vitiated pitta dośa and abnormal synthesis in the body are caused by vitiated kapha dośa and by products of abnormal activities (pathological activities) are mala dośa, they tarnish (malini karnat) the body so called mala.

In case of vāta śonita there should be abnormality in movemental activity (vāta dośa) as well as abnormality of rakta. Due to metabolic disturbances (dhātwāgni vaishamya) there is overproduction of metabolites like uric acid, calcium pyrophosphate, etc. High concentration of these metabolites in blood should be considered as raktaduśti, high concentration of uric acid and other metabolites in blood slows the propulgateion of blood in capillaries and hence there is stagnation of it near the joints and dependent parts of the body. Uric acid escapes and enters into the joints and other tissues from the stagnated hyperuricemic blood, and the same time there may be reduced excretion of uric acid through kidneys, these all disturbed movemental activities are considered as vāta duśti and over production of uric acid during metabolism of nucleoproteins and through de novo pathway may be considered as agniduśti. It is observed that patients suffering from vātaśonita also have visamāgni (disturbed digestive capability), it is a rule that if the jatharāgni (digestive capability) is disturbed the other types of agnī (metabolism) will also be disturbed. It is the beauty of Ayurvedic science that has mentioned all and very different etiological factors responsible for a) agni vaisamya b) dośa prakopa and c) dhātu dushti directly or indirectly.

Inflammation of joints may also occur by some other metabolites like calcium pyrophosphate crystal deposition which is also a by product and resultant of agni dushti (disturbed metabolism).

27 may 2016

न हि बस्तिमं किञ्चित् वातरक्त चिकित्सतम् , च .चिDue to properties ; 88/29 .  
like Sukshmatva and Saratwa of Vayu, Dravatwa and Saratwa of Rakta they spread all over the body. The spreading is facilitated by VyanaVayu. The doshas get lodged in sandhies. In this respect the control over Vayu in turn .Rakta is achieved by Basti

As Asthtidathu is involved in the disease Vatarakta, it is to be assumed that the drug acting upon pureeshadhara kala will certainly act on the Asthidara kala, as kalas of both are the same. In addition the active principles of Basti dravya administered reaches up to the grahani which is related to both Pittadharakala and majjadharakala. Hence holistic action of Basti in terms of cleansing and nourishment of Asthi dhatu, Sandhi Majja etc. and ultimately resulting in Vatashamana is perceived clinically.

The rectum has a rich blood and lymph supply and drugs can cross the rectal mucosa like other lipid membranes. The unionized and lipid soluble substances are rapidly absorbed from the rectum. In the upper portion of rectum, absorption is via the upper rectal mucosa and is carried to the superior hemorrhoidal vein into portal circulation where as that which is absorbed in the lower rectum enter directly into the systemic circulation via middle and inferior hemorrhoidal vein. This systemic assent gained is helpful for generalized cleansing followed by vigour promotion.

In the context of Vatarakta chikitsa the vitiated doshas along with mala should be expelled out by the administration of Sagritha Ksheerabasti. Here Ksheera is used to get pittahara, rakta prasadaka and Vatanulomana effect. The chemical reaction sequence originated in pakwasaya by basti passes from cell-to-cell, ultimately in to the entire body. 1/3rd of Serum uric acid is excreted through the gut and the remaining 2/3rd through the kidneys generally. Basti, because of its laxative action, increases expulsion of uric acid through gut. Even more the combined

action of Guduchi siddha yoga basti like mutrala, uricosuric properties increases the excretion of uric acid through urine.

In a nut shell, high dose administration of Guduchi (active alkaloid-Berberine) will act as analgesic, anti-inflammatory and exhibits corticosteroid action. Ksheera (Milk) used, reopens calcium channel and along with this Saindhava Lavana (Lavanat Vardhate Asthi-Yogaratnakara) enhances the integrity of Asthi dhathu. Mixture of Madhu, Saindhava, Ksheera produces Abhishyandhi guna which is influential in dissolving Urate crystals.

These observations suggest that this therapy not only produces symptomatic relief but also control the disease process and may cause long lasting relief.

स सप्तविधो अष्टादशविधो अपरिसंख्येय विधो वा भवति । चComparison of Maha ; 4/5 .नि.  
kustha with its Modern Corelation

Sr.No.

Type of Kustha

Co-relation with Modern

1.

Kapāla Kuṣṭha

Non Erythematous Eczema, Keratosis, Atrophic Actinic Keratosis, Non Hypertrophic Keratosis, Seborrhic conditions, Tuberculoid leprosy

2.

Udumbara kuṣṭha

Dyshidrosis, Discoid Eczema, Venous Eczema, Dermatitis Herpetiformis, Autoeczematization, Eczema overlaid by Viral Infections, Nodular leprosy of childhood

3.

Maṇḍala Kuṣṭha

Early and Indeterminate Leprosy, Maculo Leprosy and Lichen leprosus

4.

Ṛṣyajihvā kuṣṭha

Lyme disease, Ulcerating type and Borderline Borderline Leprosy



5.

Puṇḍarīka Kuṣṭha

Lazarine Leprosy, Erythema multiforme,

6.

Sidhma Kuṣṭha

Tinea versicolor, Pityriasis alba, Borderline Leprosy

7.

Kākaṇaka Kuṣṭha

Squamous cell carcinoma, Lepromatous Leprosy

**चर्माख्यं बहलं हस्तिचर्मवत् । च ; 21/7.चि.**

Skin is similar to elephant (pachyderms) and thick in nature , pachys means thick and derma means skin.

The main symptom is skin getting thick thus disease like scleroderma, onchocerciasis, harlequin ichthyosis etc can be considered in this group.

Building up of collagen is associated with thickening of skin. Transport of fats into the space between the skin cells may be another reason for thickening of skin.

Research shows that in diabetic patient thickness of skin is observed reason being collagen bundles become large, disorganized and separated by clear spaces. Small amount of acid mucopolysachrides may be present in upper reticular dermis. Presence of active fibroblast and extensive collagen polymerization in the rough endoplasmic reticulum may be the pathogenesis for thick skin.

Calcium deposition may also be cause for tough and thick skin. Rūkṣata, kharata, guna of vāta and shita guna of vāta and kapha alongwith increase of guruta, manda and sthira guna of kapha contribute to thickening of skin. Khara is also property of asthi dhatu which is generated by interplay between prithvi, agni and vāyu. Here calcium is representative of prithvi mahabhut. So prithvi mahabhut bhuyista āhar or increased parthivagni can lead to increase absorption of calcium from gastrointestinal tract.

Eka kushtha and ichthyosis ; अस्वेदनं महावास्तु यन्मत्स्यशकलोपमम् । तदेककुष्ठं... च.चि.7/21 आचार्य चक्रपाणि ; महावास्तु इति महास्थानम् । मत्स्यशकलोपममिति मत्स्यत्वक्सदृशम् ॥ Eka kuṣṭha , can be correlated with ichthyosis , is a skin disease caused due to vāta kapha predominancy. vāta dōṣa is responsible for the degenerative or destructive changes where as kapha is responsible for obstructive changes.

Asvēdana (Anhidrosis) may be caused by destruction of sweat glands and or integumetary system it may be due to autoimmune process or infection or anhidrosis may be due to osbtruction (kapha) in the outlet or blood supply as in microangiopathy.

Stimulation of acetylcholine and further ionic changes caused thereafter are responsible for sweating. Lack of such impulse also causes reduced sweating. Acharya charak mentions svedo ati artham in vyāna āvrita prāna ( ch.chi 28/203) , asveda in udānenāvrita vyāna (ch.chi. 28/214) .. Astanga Sangrahaakar has explained role of vyān vāyu in sweda which when hampered or becomes āvrita leads to anhidrosis , hyperhydrosis occurs by vitiated vyāna with āvrita prāna .

Anhidrosis can occur as an isolated condition or as part of a group of symptoms associated with other diseases. Anhidrosis can also occur after skin has been

injured, because sweat glands are clogged or obstructed, as an inherited defect, or as a side effect of medication.

Reduced sweating increases dryness of skin which is presented as rough, scaly or flaky skin. The word ichthyosis comes from ancient Greek where ichthys means fish. All types of ichthyosis have dry, thickened scaly or flaky skin.

Ichthyosis vulgaris is a skin condition that causes dry, dead skin cells to accumulate in patches on the surface of skin. It is also known as “fish scale disease” because the dead skin accumulates in a similar pattern to a fish scale. Another commonly accepted correlation is psoriasis.. approach to treatment is based on vāta and kapha dosha in both diseases.

31 Oct 2015

### Visarpa

**Salient features of Visarpa :** Doshah trayo malaah ; Doshaah trayo malaa iti atra dosha shabden eva vaataadi praaptau malaa iti \*ati artham dushtyaam shareera malineekaranatvam pratipaadayitum uktam .. uktam cha anyatra -Shareera dooshanaat doshaa malineekaranaat malaah , dhaaranaat dhatavah cha syuh vaata pitta kaphah trayah.. acharya chakrapani on ch.chi 21/15.. in case of visarpa , severe vitiation of tridosha occurs with involvement of rakta, laseekaa, tvak , maansa.. the vitiation of dosha is not only severe also with sudden onset and hence disease manifests acutely ; visharpanasheelaih dosheh visarpaah.. the same seven components involve in kushtha but with delayed manifestation of disease.. it shows that antibody mediated reactions are predominant in visarpa and cell mediated immunity ( T cells ) in kushtha...Marmopaghaataat ; marma iti hridayam ( acharya chakrapani on ch.chi 21/26).. visarpa if appears with full blown features may be fatal.. in this context anaphylaxis can be understood ; life threatening anaphylactic response of a sensitized human appears within minutes after systemic exposure to specific antigen and is manifested by respiratory distress due to laryngeal edema and/or intense bronchospasm , often followed by vascular collapse , or by shock without antecedent respiratory difficulty . Pruritus and urticaria with or without angioedema are characteristics. Nausea vomiting crampy abdominal pain , and diarrhea are due to GIT upset.. courtesy ; Charak samhita with chakrapani commentary and Harrison's internal medicine..

24 June 2014

Post herpetic neuralgia / Acute neuritis ; Upadravastu khalu roga uttarakaalajo rogaashrayo roga eva sthoolo anurvaa , rogaata pashchaatjaayata iti upadrava sangyah.....ch.chi 21/40.. The most debilitating complication of herpes zoster is pain associated with acute neuritis and postherpetic neuralgia...T/T... mahaavaatavidhvansa rasa or ekangaveera rasa or vrihatavaatachintaamani rasa



with dashamoola+ erandamoola+devadaaru +raasna kwaatha.. for lep same dravya of kwaatha with dashaanga lep churna + kamala+aamalaki +amritaa +yashtimadhu mixed with ghrita.....The onset of herpes zoster is heralded by pain within the dermatome , which may precede lesions by 48-72 hrs ; an erythematous maculopapular rash evolves rapidly into vesicular lesions...Harrison's principles of internal medicine (18 th edition , page no 1464).... Acharya charak mentions " agnidagdha prakaaraih cha sphotairoopachiyate, sa shighragatvaadaashveva marmaanusaari bhavati...in vaata pitta pradhaana aagneya visharpa"..ch.chi.21/36..

Vaata pitta prashamanam agni visharpena hitam.. ch.chi.21/117..

Oral antiviral therapy ; acyclovir or valacyclovir. For neuralgia ; gabapentine , amitriptyline hydrochloride , lidocaine ( patches), etc...prednisone is effective in moderate to severe pain but in restricted cases , not in presence of osteoporosis , DM, HTN . Glucocorticoids should not be used without concomitant antiviral therapy...

{

Pandu / Anaemia ; Soalparakto ch.chi.16/6; acharya chakrapani ; raktakshaya rakta poshaka rasasya pittena kshapanaad rakta poshaka saara bhaaga anutpaadaat cha... it means there is deficiency of blood cells producing nutrients/elements/vitamins like globin, iron , folic acid , cobalamine etc... in nephrotic syndrome(NS) there is loss of iron binding protein , transferrin, through urine which results in iron resistant microcytic hypochromic anaemia . In this condition punarvaadi mandur ( ch.chi.16/93-96) is effective.. as acharya charak indicates the use of this drug in paandu , shothaadi.. (edema is predominant feature in NS)

Darbha mixed goat blood basti is found effective... Hemosiderosis is due to excess blood transfusion. Iron chelating agents , raktamokshana , virechana and

raktapittahar/ raktaprasaadaka /raktashodhaka drugs (ch.su.24/18;19) are indicated

}

2 may 2013

Shishirdveshi iti sheetdveshi .chakrapani on ch.chi.16/15.....means cold intolerance due to cutaneous vasoconstriction in anaemia, a compensatory mechanism to increase blood supply to vital organs....

8 Nov 2014

### Allergy

Allergy is phenomenon where antigen antibody reaction occurs on surface of mast cells to liberate histamine and like substances to manifest vasodilatation bronchospasm edema and ulceration etc.. as per ayurved perspective there is involvement of pitta and vaata in which vaata is part of immune system and pitta as part of inflammatory peptides like histamines. So there is manifestation like shitapitta udarda kotha.. shirisha is best because of its predominance as vishaghnaanaam...The combination of shirisha tvaka , vaasaa kantakaari yashtimadhu haridra darvi tulasi amrita aamalaki pippalli is most effective.. Haridra khanda kamadudha mahamanjishthadi kvath kushthadi churna etc are indicated..

shirisho vishaghnaanaam... ch.su.25/40 it contains antihistamine...

Lipid peroxidation & उरुस्तम्भ ; स्नेहाच्चां चितं कोष्ठे वातादीन्मेदसा सह । रुद्ध्वा आशु गौरवादूरु यात्यधोगैः  
सिरादिभिः । च.चि.27/10 ; आमं चितमिति रसशेषरूपं , कोष्ठोपलेपेनाप्यामं संचितं , मेदसासहेति मेदःसहितमामं  
वातादीनां रोधकं ज्ञेयम् । आचार्य चक्रपाणि ।।

Lipid peroxidation is a free radical process involving a source of secondary free radical, which further can act as second messenger or can directly react with other biomolecule, enhancing biochemical lesions. Lipid peroxidation occurs on polysaturated fatty acid located on the cell membranes and it further proceeds with radical chain reaction. Hydroxyl radical is thought to initiate ROS and remove hydrogen atom, thus producing lipid radical and further converted into diene conjugate. Further, by addition of oxygen it forms peroxy radical; this highly reactive radical attacks another fatty acid forming lipid hydroperoxide (LOOH) and a new radical. Thus lipid peroxidation is propagated. Due to lipid peroxidation, a number of compounds are formed, for example, alkanes, malanoaldehyde, and isoprotanes. These compounds are used as markers in lipid peroxidation assay and have been verified in many diseases such as neurogenerative diseases, ischemic reperfusion injury, and diabetes. This is the impact of sama meda on the body tissue which obstructs the flow (gati) of vata leading to nutrition deficit thereby leading to damage of mamsa dhatu. Chakrapani comments mēdasā sahēti mēdaḥsahitamāmam vātādīnām rōdhakam.

Muscle tissue is unique in its requirement and ability to undertake very rapid and co-ordinated changes in energy supply and oxygen flux during contraction. Several studies have suggested that this renders the tissue particularly prone to oxygen radical-mediated damage. Free radicals have been postulated to play a role in muscle damage induced by different forms of exercise and in various pathological disorders, such as the muscular dystrophies, malignant hyperthermia and alcoholic myopathy. (Free radicals and muscle damage M J Jackson and S O' Farrell Author Affiliations Department of Medicine, University of Liverpool Liverpool, UK)

Further ama may be formed at dhatvagni and bhutagni level too which may also produce sama meda which can be further cause of disease.

Lysosomal acid lipase deficiency (or LAL deficiency or LAL-D) happens when the body does not produce enough actively lysosomal acid lipase (LAL or LIPA) enzyme. This enzyme plays an important role in breaking down fatty material (cholesterol esters and triglycerides) in the body.[1] Infants, children and adults that suffer from LAL Deficiency experience a range of serious health problems. The lack of the LAL enzyme can lead to a build-up of fatty material in a number of body organs including the liver, spleen, gut, in the wall of blood vessels and other important organs. (sirādibhiriti sirādhamaṇīsrōtōbhiḥ)

Very low levels of the LAL enzyme lead to early onset LAL Deficiency, sometimes called Wolman disease after the physician who first described it. Early onset LAL Deficiency typically affects infants in the first year of life. The accumulation of fat in the walls of the gut in early onset disease leads to serious digestive problems including malabsorption, a condition in which the gut fails to absorb nutrients and calories from food. Because of these digestive complications, affected infants usually fail to grow and gain weight at the expected rate for their age (failure to thrive). As the disease progresses, other complications develop including increasing liver dysfunction or liver failure.

People who are older children or adults generally present with a wide range of signs and symptoms that overlap with other disorders. They may have diarrhoea, stomach pain, vomiting, or poor growth, a sign of malabsorption. They may have signs of bile duct problems, like itchiness, jaundice, pale stool, or dark urine. Their feces may be excessively greasy. They often have an enlarged liver, liver disease, and may have yellowish deposits of fat underneath the skin, usually around their eyelids. The disease is often undiagnosed in adults.

23 May 2016



Acharya chakrapani and modern science approach to hydrotherapy / swimming -  
the Physical Effects of Hydrotherapy on the Body

Hydrotherapy uses water to deliver temperature and pressure changes to the body. These changes are sensed by the body via nerve endings in the skin and muscle, and result in neural “reflex effects” that are controlled by the brain and spinal chord. The most important of these reflex effects are vasodilatation and vasoconstriction, which are the terms used to describe the relaxation and tensing of the blood vessels in the body. These physical changes in the blood vessels cause changes in the rate of blood flow and in the metabolic functions that are linked to the rate of blood flow.

Which changes happen in the body are dependent on the outside stimuli it receives; whether the water is hot or cold, the motion of the pressure, and the strength of the pressure, too.

Cold water or ice has, in essence, an opposite effect on the body than hot water. Cold water and ice causes the body to try and conserve heat. As a result, blood vessels in the body constrict, decreasing the amount of blood that flows through them. Blood flow is diverted from the extremities to the core of the body and to internal organs, to help keep them warm and operating correctly. The pores of the skin close, sweat glands shut down, muscles tense, and some endocrine system organs, like the adrenal gland, become more active.

Over short durations, cold water makes a person more alert and makes them feel less tired as the body activates these neural networks that work to create heat and raise blood pressure in response to the cold. (jalēna bahirnirgacchadūṣmaṇō niruddhasyāntaḥpravēśācchlēṣma saṅghātabhēdanam bhavati, tathā pratarāṇakriyayā ślēṣmā vicchidyatē, samānābhimatā kriyā'pyatra yōgyatvāt kriyata ēva , जलेन बहिर्निर्गच्छदूष्मणो निरुद्धस्यान्तःप्रवेशात् श्लेष्म संघात भेदनं भवति , तथा प्रतरण क्रियया श्लेष्मा विच्छिद्यते , समानाभिमता क्रिया अपि अत्र

योग्यत्वात् क्रियत एव . acharya chakrapani on C. Chi. 27/ 59). The great vision of our Maharshi created the Ayurveda effective in all aspects of health and disease..

Prataarayet pratisroto nadeem sheetajalaam shivaam. Sarah chaa vimalam sheetam shthira toyam punah punah.. ch.chi 27/59. Hydrotherapy is very effective in reducing spasticity of cerebral palsy , spinal injury and upper motor neuron diseases.. its also effective in all types of myopathy and neuromuscular diseases ; uroostambha, ekaangaghaata, pakshaaghaata, sarvaangaghaata , snaayugata vaata , maansamedogata vaata etc.. i have seen results of hydrotherapy in above mentioned diseases.. Trio of panchakarm , physiotherapy and hydrotherapy are best available therapies for neuromuscular diseases.

**Schindler disease**, also known as Kanzaki disease and alpha-N-acetylgalactosaminidase deficiency is a rare congenital metabolic disorder in humans. This lysosomal storage disorder is caused by a deficiency in the enzyme alpha-NAGA (alpha-N-acetylgalactosaminidase), attributable to mutations in the NAGA gene on chromosome 22,[1][2] which leads to excessive lysosomal accumulation of glycoproteins.[3] A deficiency of the alpha-NAGA enzyme leads to an accumulation of glycosphingolipids throughout the body. This accumulation of sugars gives rise to the clinical features associated with this disorder. Schindler disease is an autosomal recessive disorder, meaning that one must inherit an abnormal allele from both parents in order to have the disease.

There are three main types of the disease each with its own distinctive symptoms.

In the Type I infantile form, infants will develop normally until about a year old. At this time, the affected infant will begin to lose previously acquired skills involving the coordination of physical and mental behaviors. Additional neurological and neuromuscular symptoms such as diminished muscle tone, weakness, involuntary

rapid eye movements, vision loss, and seizures may become present. With time, the symptoms worsen and children affected with this disorder will experience a decreased ability to move certain muscles due to muscle rigidity. The ability to respond to external stimuli will also decrease. Other symptoms include neuroaxonal dystrophy from birth, discoloration of skin, Telangiectasia or widening of blood vessels.

In Type II adult form, symptoms are milder and may not appear until the individual is in his or her 30s. Angiokeratomas, an increased coarsening of facial features, and mild intellectual impairment are likely symptoms.

Lipid storage disorders (or lipidoses) are a group of inherited metabolic disorders in which harmful amounts of lipids (fats) accumulate in some of the body's cells and tissues. People with these disorders either do not produce enough of one of the enzymes needed to metabolize lipids or they produce enzymes that do not work properly. Over time, this excessive storage of fats can cause permanent cellular and tissue damage, particularly in the brain, peripheral nervous system, liver, spleen and bone marrow.

Lipid Myopathies: Clinical classification

Fixed weakness

OCTN2: Carnitine transporter

Riboflavin-responsive MAD deficiency (ETFDH)

SCAD

MCAD

Multiple acyl-CoA dehydrogenase deficiency (GA II) (MADD)

Neutral lipid storage disease with ichthyosis (NLSDI)

Neutral lipid storage disease with myopathy

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Exercise intolerance, Cramps & Myoglobinuria

CPT II deficiency

VLCAD

Short-chain 3-hydroxyacyl-CoA dehydrogenase deficiency (HADH)

Trifunctional enzyme deficiencies (MTP) A & B

MCKAT

Phosphatidic acid phosphatase (LIPIN 1)..



**My approach why udaavarta is mentioned in Trimarmiya chikitsa  
adhyaya of charak samhita**

It explains the treatment of diseases effecting three vital systems (cerebrovascular, cardiovascular and renovascular system) and not the organs themselves.

Brain, heart and kidney axis support each other in maintaining the homeostasis. Declining cardiac function is associated with a spectrum of compensatory mechanisms to preserve cardiovascular homeostasis. Two of the major participants in the neurohormonal system that are intricately intertwined in order to achieve stability are (i) the Autonomic nervous system and (ii) the Renin–angiotensin–aldosterone system (RAAS). A reduction in cardiac output activates afferent stimuli from the baroreceptors to the central nervous system cardio-regulatory centres, which in turn leads to an activation of the sympathetic nervous pathway. Reduced renal perfusion, secondary to reduced forward flow activates the RAAS system via renin release. Importantly, renin facilitates the conversion of angiotensinogen to angiotensin I. Angiotensin-converting enzyme subsequently converts angiotensin I to angiotensin II. Although angiotensin II has a central effect on increasing sympathetic activity, it is also involved in sodium and water retention and has a systemic vasoconstrictive effect. It is noteworthy that these compensatory mechanisms are initially important to maintain cardiac output but over the long term are detrimental through their adverse impact on the structural adaptive response of the heart. Heightened sympathetic tone modulates heart rate, enhances AV conduction, as well as myocardial contractility, but when sustained over time it is associated with reduced cardiac sympathetic neuronal density and responsiveness. Sympathetic activation in turn increases the vasoconstrictor tone, accompanied by activation of the RAAS and the endothelin 1 and vasopressin system, which may be responsible for peripheral organ dysfunction and damage in the setting of congestive heart failure.

Thus a functional interrelationship is essential for homeostasis as well as disease condition. Diseases such as diabetes mellitus have an impact on all the three system and they in turn have impact on each other. Similarly, Acharya Charak observed Udavarta, a disease of gastrointestinal tract origin and dominated by vata doṣa to have impact on this trimarma. In other words, these Marma have to be protected especially from Anila (vāta), as vāta is the main cause for the aggravation of Pitta and Kapha and also is the cause of Prāna (Life force).

Vāta gets vitiated due to the vātaja aggravating factors and especially retaining or unnecessarily provoking the natural urges. Caraka in Vimānasthāna mentioned that diet if consumed without following the rules of proper dietary intake can lead to manifestation of disease by vitiating doṣa and deteriorating the healthy status of Dhātu.

The toxins retained due to retention of mala due to impaired apana can lead to various diseases. The impaired apana has first impact on agnito hamper the metabolism. The impaired metabolism leads to impaired gut microbiome and the latest research has shown that impaired gut microbiota can lead to various disorders, from heart disorders to psychological disorders which the Acharya have mentioned in verse 9-10.

It is generally observed that patient exaggerate pressure to evacuate the faecal matter which is not easily passed in case of udavarta. This increases the intra rectal pressure which can lead to arsha (piles), parikartika (fissure). The continuous increased pressure can further lead to bleeding leading to anaemia (pandu). As discussed in shwas adhyaya, pandu is cause of shwas (dyspnoea). Shwas has its impact on hridaya (heart) as it is seat of pranvahasrotas. Hridaya may also get involved due to pandu. Involvement of hridaya opens the gate for various disorders from brain to kidneys. Increased rectal pressure further leads to increased abdominal pressure which has been recorded as cause for TIA/ CVA in elderly individuals. Increased abdominal pressure has impact on movement of diaphragm which further increased the thoracic pressure. Restlessness attained

due to improper evacuation of faeces increases the irritability and non attentiveness leading to psychological distress. Some Acharyas have accepted guda as a sthana of mana. (6-10)

It is also observed that due to impaired digestion and absorption, nutritional deficiency occurs especially folic acid etc which lead to increased homocystine levels, another cause for various serious diseases of three marma.

Therefore, one finds explanation of Udavarta which as a disease alone only has impact of abdominal discomfort but later on may be the basic pathogenic factor for various diseases related to three marma. Hence Caraka explained Udavarta as a disease of gastrointestinal tract prior to the explanation of diseases like mutrakrichra, hridroga and shiroroga.

#### 1. Pathophysiology of Udavarta and its complication

> Food predominant in Kashaya, Katu, Tikta Rasa>

Ruksha bhavad (Ca. Su. 26/60)

> Katu Vipaka (Ca. Su. 26/58)

> Increase of vitiated vata and Increase ruksha guna>

Impaired peristalsis and increased absorption of fluid content as a feature of vikarvighat bhava>

Difficulty in passing/ retention of mutra (urine), purisha (faeces), vata (flatus) and retasa>

Further vitiation of movement of vata>

If takes tiryak gati > Gulma

Vitiated vata if takes upward direction>

Further hampers digestion process > Reduced nutrition

Retention of toxic factors > Leads to ama visha>

Dushit aahar rasa Nutritional Disorders (aandhya, badhirya)>

Ama janya vikar depending on sthana like hridroga, rajyakshma, mutragata etc (Ca. Ci 15/)



## **Ashmari bhavan ; ayurved and modern approach**

Nephrolithiasis, or kidney stone, is the presence of renal calculi caused by a disruption in the balance between solubility and precipitation of salts in the urinary tract and in the kidneys. In other words there is disruption of ratio between the parthiv and jaliya concentration. Either there is increase in parthiv substrates or decrease in jaliya (fluid) concentration or both.

Kidney stones develop when urine becomes "supersaturated" with insoluble compounds (parthiv) containing calcium, oxalate ( $\text{CaOx}$ ), and phosphate ( $\text{CaPO}_4$ ), resulting from dehydration or a genetic predisposition to over-excrete these ions in the urine.<sup>25</sup> Modern lifestyle, dietary habits and obesity emerge to be the promoters of idiopathic stone disease. Flesh of animals of marshy area, fish, and indulgence in overeating are the aetiological factors mentioned for madhumeha, sthaulya and ashmari too.

High protein diet: From a urinary point of view, it has been demonstrated a long time ago that the main effect of a high protein intake is a rise in urinary calcium excretion, independent from other dietary factors such as salt intake. Dietary proteins, especially of animal origin (Flesh of animals of marshy area, fish), actually lead to a high potential renal acid load (PRAL), and decrease in urinary pH and a state of mild chronic metabolic acidosis. There are also other urinary factors driving the risk for kidney stones in high protein diets. For example, a significant reduction in urinary citrate levels has been demonstrated. These modifications may be due to the lower content of citrate in diet, since high protein diets usually do not include large amounts of fruit and vegetables. Moreover, a habitual high dietary intake of sucrose is associated to a high risk of kidney stone onset. Further high purine intake diet is cause for hyperuricemia which further leads to uricosuria a cause for dysuria.<sup>26</sup>

Excessive salt intake: The strong connection between salt intake and nephrolithiasis has also been demonstrated by Curhan & coll. in a large

epidemiologic study on healthy middle-aged women, highlighting a higher risk for calcium stone onset or recurrence in those who have a daily salt intake in the highest quintile. If the patient continues to consume a high sodium diet, sodium will reach the distal nephron and increase the excretion of calcium and potassium along with citrate, resulting in a change in the urinary pH that will eventually increase the risk of stone formation.<sup>27</sup>

Vegetarian diet has been recognized as protective against kidney stone disease. Fruit and vegetables, the main components of vegetarian diets, actually have a low content in proteins and sodium chloride and a high content in lithogenesis inhibitors such as magnesium, citrate and alkaline potassium.<sup>28</sup>

High intakes of potassium and phytate, which are considered reliable indexes of fruit and vegetables consumption, are actually associated with lower risk of incidence of nephrolithiasis in groups with different age and sex.<sup>29</sup>

But Dietary oxalate may be important in stone development; spinach, beets and rhubarb in particular, contain large amounts of oxalate and they may increase urinary oxalate excretion and predispose to the development of calcium oxalate stones.<sup>30</sup>

#### Milk and milk products

These findings have also been confirmed by the large epidemiologic studies carried out by Curhan & coll, showing that people in the highest quintile of milk and dairy product consumption are at the lowest risk for kidney stone onset.<sup>31</sup>

Anyway, a dietary approach with low salt, low animal protein and high fruit and vegetable intake, together with a normal and balanced consumption of milk, dairy products, carbohydrates and fats, is the best way to prevent kidney stone and relapse at the current state of knowledge. Phytates are present in whole grains and legumes and they can inhibit CaOx stone formation.

Decreasing the consumption of meat, chicken and seafood will decrease the intake of purine and, therefore, production of uric acid. Higher intake of fruits and vegetables should raise urinary pH and reduce the risk of uric acid crystal formation.<sup>32</sup>

Intestinal hyperoxiluria is best avoided by restricting cocoa drinks, chocolate, candies, black tea, excessive coffee intake, spinach rhubarb, asparagus, celery, parsley and tomatoes. Calcium oxalate stone formers should limit intake of almonds, peanuts, cashews walnuts, beetroot, cheeko, cocoa, chocolate, tomato, strawberries, eggplant, soy products, wheat bran and rice bran.<sup>33</sup>

Treatment of ashmari (calculus):

Pashanbheda: Pashan- Litho Bheda- tripsy.

Treatment should be directed in multiple directions. Drugs such as pashanbheda, varun, gokshura, haritaki which have impact of breaking the calculi. Drugs like punarnava, dasamula, ushira, sariva, helps in flushing out the crystals. Shatavari, sariva, sneha helps in preventing haematuria.

3 May 2013

Kshine sarakt mutratvam parshv prishth kati grah.ch.ch.11/13..... Renal Tuberculosis...

7 April 2014

Nephrolithogenesis... urinary stones usually arise because of the breakdown of a delicate balance between solubility and precipitation of salts. When urine becomes supersaturated with insoluble materials, because excretion rates are excessive and/or because water conservation is extreme, crystals form and may grow and aggregate to form a stone... Acharya chakrapani ;



Vaatajaayaamashmaryam prabalen vaayunaa mootrashoshe kriyamaane tadgatah kaph ev ashmari roopah kriyate.. on ch.ch.26/36. There is imbalance between ap and prithvi mahaabhoot in urine , either increase prithvi mahaabhoot or decrease ap mahaabhoot or both lead to lithogenesis.. T/T ; Hydration therapy and mootral drugs to increase ap mahaabhoot in urine and pakshaanbhed like drug to breakdown stone in small quantity to be flushed through urine.. pakshaan means stone or litho and bheda means tripsy or fragmentation of stone ; lithotripsy , is done with shock waves ( extracorporeal lithotripsy ) , is interchangeable with pakshaanbhed ( A drug)..!! My choice of combination of drugs is punarnava , gokshuru , varun , pakshaanbhed , shatavari and haritaki....

Acharya charak stated : tasya lingam ajirnayasya ..... Mootrarogaam cha mootrastham ... Ch.chi.15/ 45-49. When annavisha affects the mootra ( urine / urinary tract ) manifest various urinary disorders/ renal diseases.. in ch.chi 26 , he mentioned mootrasanga and bastishotha as clinical features of udaavarta , again , he referred ajeerna as one of causative factor of mootrakrichchha..

Modern time observations:

Between the large intestine and the kidney, a bi-directional functional relationship exists. Uremia influences the colonic microbial metabolism whereas microbial-related metabolites are involved in the progression of the kidney diseases . p-Cresyl sulphate and indoxyl sulphate have been most extensively studied and are considered as prototypes of the so-called uremic toxins. They are derived from bacterial fermentation of the aromatic amino acids tyrosine and tryptophan, respectively, followed by sulphation in the colonic mucosa or the liver. Within the plasma, they are highly protein-bound and accumulate when kidney function fails. The free, unbound levels of these solutes increase more than their total plasma levels due to competition for binding sites on plasma proteins. In patients with chronic kidney disease, both p-cresyl sulphate and indoxyl sulphate levels have been linked to overall mortality, CVD and progression of the kidney disease .



Acharya charak : vinshati vidhāh krimayah pūrvamuddishtā nānāvidhen pravibhāgen anyatra \* sahajebhyah \* .. vi.7/9 .

acharya chakrapani : anyatra sahajebhyah iti anen \*\*sharira sahajāh tu avaikārikāh krimayo \*\* vinshati api adhikā bhavanti iti darshayati..

Modern time observations : The human microbiota is the aggregate of microorganisms that resides on or within any of a number of human tissues and biofluids, including the skin, mammary glands, placenta, seminal fluid, uterus, ovarian follicles, lung, saliva, oral mucosa, conjunctiva, biliary and gastrointestinal tracts. They include bacteria, archaea, protists, fungi and viruses. Though micro-animals also live on the human body, they are typically excluded from this definition. The human microbiome refers specifically to the collective genomes of resident microorganisms.

Humans are colonized by many microorganisms; the traditional estimate is that the average human body is inhabited by ten times as many non-human cells as human cells, but more recent estimates have lowered that ratio to 3:1 or even to approximately the same number. Some microbiota that colonize humans are commensal, meaning they co-exist without harming humans; others have a mutualistic relationship with their human hosts. Conversely, some non-pathogenic microbiota can harm human hosts via the metabolites they produce, like trimethylamin. Certain microbiota perform tasks that are known to be useful to the human host; the role of most resident microorganisms is not well understood. Those that are expected to be present, and that under normal circumstances do not cause disease, are sometimes deemed normal flora or normal microbiota.

**Mutrakrcchra (Dysuria) is of eight types which are as follows**

1-4. Dysuria is caused due to aggravated vāta, pitta, and kapha individually, and all the doṣa aggravated simultaneously (sannipatika) , 5 . Dysuria is caused due to calculus in the urinary tract.

6. Dysuria is caused due to sarkara (granules).

7. Dysuria is caused due to diseases of semen.

8. Dysuria is caused due to kshata (trauma) to urinary tract.ver.

Significance of vata doṣa in normalcy and disease condition has been mentioned in Ca. Su. 12 and Ca. Ci. 28 but considering the disease affecting the three marma, vata needs to be understood for various somatic diseases. Vata is the primary doṣa in udavarta and later complicates into various psycho-somatic disorders. Similarly, in urine formation vata doṣa has significant role which may be understood as follows. Vyan vayu is the one which regulates the cardiac output thus maintaining the systolic pressure in the renal vessel. Saman vayu maintains the electrolyte and chemical balance by absorbing and filtering the electrolytes. The hydrostatic pressure gradient across the glomerular capillary wall is the primary driving force for glomerular filtration. Oncotic pressure within the capillary lumen, determined by the concentration of unfiltered plasma proteins, partially offsets the hydrostatic pressure gradient and opposes filtration. As the oncotic pressure rises along the length of the glomerular capillary, the driving force for filtration falls to zero before reaching the efferent arteriole. Several factors, mostly hemodynamic, contribute to the regulation of filtration under physiologic conditions. The energy required at cellular level is generated by converting the ADP to ATP and this is coordinated by udan vayu which is the stimulant (pravrittimulak) whereas Pran vayu is the controller as well as it is the receptor which understands the acid base balance within the blood. Autoregulation of glomerular filtration is the result of autonomous vasoreactive

(myogenic) reflex in the afferent arteriole which is brought about by pran vayu. Lastly apan vayu has the role to help excretion. The action of apan is seen at the distal collecting duct level.

Susruta describes Pittaja and Kaphaja types of Mutraukasada as two different conditions. In Pittaja Mutraukasada he describes that on drying, the urine resembles Gorocana curna and in case of Kaphaja variety, on drying the urine becomes like Sankha curna.

Asmari (calculus) is of four type Vataja, pittaja, Kaphaja, sukraja.

Withholding the urge to urinate causes vāta to get obstructed in its normal pathway which causes Udāvarta (reversal of movements) and thereby the Mutra fills up in the Udara producing inconsistent pain, sense of indigestion, obstruction to the flow of Mutra.

Indulgence in sexual intercourse by a person with active urge to micturate causes affliction of the dislodged Sukra which flows either before or after the urine stream and this is called MutraKrcchra.

Mutrakriccha is defined as a condition where an individual finds it difficult to pass urine and it is associated with severe pain. The cause may be understood at two levels i) at the level of glomerular filtration or formation of urine and ii) at the level of the passage of urine i.e. in calices of kidneys, ureters, bladder, urethra, penis/ vagina.

Various types of crystals which pass through the glomerular filtration form an important cause for irritation of urinary tract cells. Crystals of uric acid, calcium oxalate, phosphate contribute to difficulty in passing urine. Increase acidic pH of urine also causes painfull and difficulty in passing urine.

Urinary tract infection: In the majority of UTIs, bacteria establish infection by ascending from the urethra to the bladder. Continuing ascent up the ureter to the kidney is the pathway for most renal parenchymal infections. Any foreign body in

the urinary tract, such as a urinary catheter or stone, provides an inert surface for bacterial colonization. Abnormal micturition and/or significant residual urine volume promotes true infection. In the simplest of terms, anything that increases the likelihood of bacteria entering the bladder and staying there increases the risk of UTI. Bacteria can also gain access to the urinary tract through the bloodstream.

In women, vaginal ecology is an important environmental factor affecting the risk of UTI. Colonization of the vaginal introitus and periurethral area with organisms from the intestinal flora (usually *E. coli*) is the critical initial step in the pathogenesis of UTI. Sexual intercourse is associated with an increased risk of vaginal colonization with *E. coli* and thereby increases the risk of UTI. Nonoxynol-9 in spermicide is toxic to the normal vaginal microflora and thus is likewise associated with an increased risk of *E. coli* vaginal colonization and bacteriuria. Any condition that permits urinary stasis (vegadharan) or obstruction predisposes the individual to UTI. Foreign bodies such as stones or urinary catheters provide an inert surface for bacterial colonization and formation of a persistent biofilm. Thus, vesicoureteral reflux, ureteral obstruction secondary to prostatic hypertrophy, neurogenic bladder, and urinary diversion surgery create an environment favorable to UTI.



## My approach to vāta vyādhī (Ch. Chi. 28) -

### Introduction

A careful, critical and unbiased study of the classical Ayurvedic texts show that by the time the samhita granthas were compiled, the Science and Art of Ayurveda had already passed through the stage of specialization and, knowledge flowing from different specialized fields of medicine and allied sciences generalized, simplified and principles enunciated. It is thus seen that, even as early as the time of Punarvasu Atreya and Dhanwantri, many concepts, such as: Man is a composite whole of matter, mind and spirit; he is part of much large universe in which he lives and a creature of a relatively smaller environment that surrounds him; the larger universe and his immediate local environment- the physical, , biological and psychological- act on him as stressors and he reacts to them with suitable responses engendered by internal stresses, he is constantly call upon to adapt and condition his internal environment suitably in keeping with the ever changing vicissitudes of his external environment and maintain a steady state equilibrium- dosha, dhatu mala samyata; his internal steady state represents an unstable equilibrium of the three fold forces and factors that govern all vital events and processes vata pitta and kapha- which tend to become disturbed- dosha vaisamya- under the influence of (a) internal stressors which may be somatic or psychic in nature and origin i.e. adhyatmika or (b) stressors that operate on him from his external environment i.e. adhbhautika and (c) others that are due to providential dispensation and on which man has no control i.e. adhidaivika. Asatmendriyarthā samyoga, parinama and pragnaparardha are the only three causes of disease; disease is a process and not a state- the process once initiated, moves through six consecutive evolutive steps also known as shatkriyakala.

The exciting factors of the disease are many and varied but the actual intrinsic factors which become excited and imbalanced, either conferring a predisposition

to or actually causing morbidities. The intrinsic factors as per Ayurved are the Tridosha. These are Vata, Pitta and Kapha. These three factors are so called as they are susceptible to imbalance and vitiation. In their turn, they vitiate other structural and functional elements of the living body, known as the dhatus and mala.

The Ayurvedic concepts of physiology, pathology, diagnosis, prognosis, medicine, and therapeutics are all based on the doctrine of tridosha. The tridosha predispose the body to disease when their equilibrium is disturbed and contribute to healthy state when their equilibrium is undisturbed. In other words, the normalcy of tridosha corresponds to physiological state and the imbalance represents the pathological state. All the function of the body is controlled by three fundamental factor called Tridosha. As per Ayurveda they are the pillar of the body. They all in equilibrium and located in their places perform their normal functions by which the body is sustained free from disorders. If they move on wrong path or are unbalanced they afflict the body with disorders relating to their location and functions and take away life shortly.

Among these three dosha, Vata has very much significance. It plays a key role behind maintenance of body in normal healthy state. Vata is strongest of all dosha and it causes large number of diseases. It also causes emergency condition.

The term 'vāta' is derived grammatically by the application of 'tan' pratyaya (suffix) and expelling 'n' pratyaya later to the Sanskrit verb root 'vā' which means 'gati gandhanayoh' or by the application of 'kta' or 'krt pratyaya' to the verb root 'vā' which means the same as above. The term vāta is derived from 'vātiti vāta', The verb root 'vā' means gati and gandhana. The meaning of 'gati' is 'prapti'(to acquire); and jñāna (to get aware or to sense). The term 'gandhana' means either utsāha, or preranā (to enthuse, to excite or to stimulate). Considering the different meanings of gati, and gandhana it is understood that the term 'vāta' itself conveys its role as a receptor as well as stimulator. Hence it can be said that

vāta is the biological force which recognize and stimulate all the activities in the body.

Vāta is the prime dosha explained in Ayurveda. When non vitiated Vāyu is at its abode with unobstructed movement, is responsible for lengthy 100 years of life devoid of diseases.

Vāta performs all its activity for a healthy long life provisional to its three functional statuses. They are 'akupita', 'sthānastha' and 'avyāhatagati'. Akupita means neither increased or decreased nor vitiated. It is otherwise called as 'sama' or optimal (qualitatively and quantitatively). Even if vāta is optimal in amount it should be located in its own said position like prāna in vertex, udāna in thorax etc. Then it is called as 'sthānastha', if they are dislodged from its abode and get located in an odd place (eg. Vāta get located in āmashaya) than it causes disease. Even if vāta is optimal and stay at its own site, nothing should interfere with its movement, so called as 'avyāhatagati'. 'Gati' is a characteristic feature of vāta. Gati is nothing other than directional aspect of 'cala' property. Prāna is located in vertex and has a gati towards thorax, trachea, tongue and nose. If anything obstructs this gati it leads to disease. These three characteristics of vāta imply three possible mode of pathogenesis in vāta diseases. These are svātantra dushti, gata vāta and āvarana. Further, due to the following three important properties of vāta, it is regarded entirely different from other dosha.

Asamghāta (Incorporeal)

Anavasthita (Unstable)

Anāsādhya (Inaccessible)

Pitta and Kapha have appendages and are relatively compact. On the contrary vāta is incorporeal (avayavasamghātarahita). It can be termed as rarified in nature. The vāta is anavasthita (unstable) too. These two properties are due to its panchbhautik composition. Vāta is formed by Ākāsa and Vāyu predominantly.



Ākāsa and Vāyu are incorporeal (Amurta). Calatva (mobility) and Apratighāta (unobstructability) are characteristics of Vāyu and Ākāsa perceptible by the tactile sense organ. According to Tarka Samgraha, Vāyu is devoid of shape (ruparahita) and possess sensibility to touch (sparsāvān). The biological vāta (which is present in the living being) is self originated (svayambhu), subtle (sukshma) and all pervasive (sarvagata). It is not sensible (avyakta) but its activities are patent or manifest (vyaktakarma). Anavasthita is due to cala property of vāta. This continuous moving nature of vāta is explained with other terminologies also like seeghravat (swift movement), āsukāri (instantaneous action), muhuscāri (rhythmic movement). It abounds in the fundamental quality of raja (the principle of cohesion and action). The predominance of rāja is responsible for the instability of vāta. The quality of calatva is directional in nature, which is explained by the term gati. Vāta convenes all bodily activities by this important feature.

Owing to its incorporeal nature and instability vāta is anāsādhyā (inaccessible) also. The inaccessibility is characterized in regard to its functional and physical attributes but more relevant regarding the therapeutic aspect. Above explained cardinal features make vāta acintyaveerya (inconceivable prowess) and dōshanām netā (propeller of all functional elements in the body).

Vāta as explained earlier is the biological force present in the body which recognizes and stimulates all the activities. Instability of vāta makes it inaccessible. It is characterized by an increase in the 'cala' property, which is favored and contributed by other properties also. The 'cala guna' is directional in nature and then termed as 'gati'. 'Gati' is the distinct quality of vāta, very important on physiological and pathological aspects. The gati of individual components of vāta is to be analyzed according to the intensity, direction and area; depends upon the particular function it is carrying out. When the gati is aggravated (gatatva) or obstructed (āvarana) the functional normality's of vāta are impaired. The gati or gatatva have two implications, one subjected to activity (to move, carry out or reach-gata) and the second subjected to abode of activity (pathway). Gatatva is



an essential part of any vātaja samprapti. Gatatva of vāta is possible in dhātu, upadhātu, āsaya, avayava, etc. Consumption of 'āhar' of relatively higher 'kittānsa leads to diminution of dhātu and aggravation of vāta. It leads to riktatā and more avakāsa in dhātu, makes the engorgement and hyper movement of aggravated vāta in the site. Dhātugatavāta and dhātuāvrta vāta are also distinct pathologies as in āvarana the vitiation of vāta is passive and the gati is obstructed.

In Ca.Su.12 vātakalākaliya adhyāya question regarding qualities of vāta, its exciting and alleviating factors have been raised. Rūkṣa, laghu, śītā, dārūna, khara and vishada have been explained as qualities of vāta. Repeated use of these qualities, such like substances and actions of such similar qualities causes vridhi of vāta and excited vāta is alleviated by use of substances possessing contrary qualities. The questions and answers extracted above bring out a clear contrast, two mutually interrelated and inseparable aspects of the phenomenon of sharira vāyu viz. (1) that, the sharira vāyu is a biophysical force and (2) that it is closely associated with material substances which form part of the structure of the body or, in other words, like the nervous phenomenon, it is a chemical reaction sequence which occurs during the course of life processes. It will follow from this that, this chemical reaction –sequence, can be accelerated (excited) or inhibited by homologous substances (dravya sāmānya), qualities (guna sāmānya) and actions (karma sāmānya) or the opposite of them, respectively. In other words, it may be concluded that the bio – physical force – the sharira vāyu – is closely linked with some material structural factors of the body which are susceptible of being influenced by substances āhara and aushada – homologous to these structural entities or which may possess homologous properties or actions and, in the reverse direction, the opposite of these, produce contrary effects.

Sodium, Potassium, Calcium, Chloride, ions are continuously moving around [Brownian movement] which is the result of its ionic state [swabhāva / swayambu guna]. The ionic inflow and outflow within the cell causes depolarization and repolarization or in other words impulse is generated. Hyper or Hypo state of this

ions is the cause for disease condition which may be understood in the form of seizures, palpitations, muscle cramps, lethargy, altered sensorium, coma and death.

Therefore the biological energy produced by this ionic movement is the cause for sharira vāyu and as per modern science too their concentration depends on āhara and vihara.

The continuous, controlled movement of the ions is responsible for cell activity which together at the level of cells contributes to tissue activity which together contributes to the organ, system and in turn whole body.

The functional status of Vāta with its sub units can be better understood by analyzing certain physiological events. The normal electrical conduction in the heart allows the impulse that is generated by the sinoatrial node (SA node) of the heart to be propagated to, and stimulate, the cardiac muscle (myocardium). The myocardium contracts after stimulation. It is the ordered, rhythmic stimulation of the myocardium during the cardiac cycle that allows efficient contraction of the heart, thereby allowing blood to be pumped throughout the body. Signals arising in the SA node (located in the right atrium) stimulate the atria to contract and travel to the AV node, which is located in the interatrial septum. After a delay, the stimulus diverges and is conducted through the left and right Bundle of His to the respective Purkinje fibers for each side of the heart, as well as to the endocardium at the apex of the heart, then finally to the ventricular epicardium.

On the microscopic level, the wave of depolarization propagates to adjacent cells via gap junctions located on the intercalated disk. The heart is a functional syncytium (not to be confused with a true "syncytium" in which all the cells are fused together, sharing the same plasma membrane as in skeletal muscle). In a functional syncytium, electrical impulses propagate freely between cells in every direction, so that the myocardium functions as a single contractile unit. This is the avyāhata gati of vāta which is necessary for the rapid, synchronous depolarization

of the myocardium. Conduction from SA to AV to bundles and Purkinje fiber is the *aparityakta swa mārṅa* of *vāta*. This rhythmical and conductive system of the heart is susceptible to damage by heart disease, especially by ischemia of the heart tissues resulting from poor coronary blood flow. The result is often a bizarre heart rhythm or abnormal sequence of contraction of the heart chambers, and the pumping effectiveness of the heart often is affected severely, even to the extent of causing death. This explains the *vyāhata gati* of *vāta* which is the cause of death.

The circulatory system is the main method for blood transportation within body. This system is a complex highway of vessels, and its main purpose is to move blood and nutrients throughout body. The circulatory system is also responsible for exchanging gases and removing waste products from body. Unlike an open circulatory system, a closed circulatory system is more structured and controlled. The blood of a closed system always flows inside vessels. These vessels make up the plumbing circuit of the body and can be found throughout the entire body. This plumbing circuit can be broken down into three different types of vessels, or tubes that transport blood throughout the body: arteries, capillaries and veins. Thus a continuous flow of blood from Left ventricles to the aorta to arteries all over the body then to arterioles into capillaries into venules into veins and back to the right atrium then right ventricle via pulmonary artery to the lungs and via pulmonary veins to the left atrium and back to left ventricle. This is how blood is propagated from heart to the periphery and back to the heart. The modern explanation resembles Caraka explanation as mentioned in *Ca. Ci. 15/36*

This function of *vāta* is *swa sthānastha* which helps to maintain the homeostasis or *swāsthya* but when *avarodh* to this *gati* takes place may be due to any reason the *swa mārṅāsthita vāta* gets *vimārṅa gata* as explained in *samprapti* of *śōtha* (*Ca. Ci. 12/8*).

Various edemas are either due to excessive secretion (*apāna vāyu*) or reduced absorption (*prāna vāyu*) as understood in *samprapti* of *udara*. Disturbed

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concentration of solutes and solvents causes changes in pressure (vyāna vāyu) either intravascular or extra vascular. The electrolyte balance is brought about by sweda dōṣa ambu srotas sthāyi vāyu i.e. samāna vāyu.

Prakruti sthita vāta is the one which is akshina vridha: Reduced respiratory rate due to depressed respiratory centre explains kshina prāna vāyu whereas vridha prāna vāyu may be one of the causes for increased ventilation.

Prayatna, urjā are functions of udāna vāyu. Excessive excitation of cell due to excess action potential explains the vridha udāna vāyu whereas inhibition of cell activity due to reduced action potential is due to kshina udāna vāyu.

Excessive stimulation of agni (atyagni) causes increased appetite one reason being vridha samāna vāta whereas agnimāndya, grahani etc may be caused by decrease stimulation of agni by samāna vāta.

Normal pulse rate ranges from 60-80/min. Excessive pulse rate explains the repeated contraction of heart one of the cause being excessive ākunchan prasārana karma of vyāna vridhi whereas one of the cause of bradycardia may be kshina vyāna vāyu.

Increased peristalsis is the cause for increased frequency of stools one of the reason being vridha apāna vāta whereas reduced peristalsis causes constipation one reason being kshina apāna vāta.

Various types of manifestation of the disease of vata are being explained. They include the nanatmaja vikara or individual vata prakopa, anubandha or associated vata prakopa, gata vata or accumulation of vata in dhatu or mala and Avarana. They all are having different etiopathogenic mechanisms. Avarana is one of the most complicated basic fundamental concepts of Ayurveda. It is a unique as well. To get one understood about Avarana, the basic principles are to be dealt with in detail.



25 May 2016

My approach, to Certain etiological factors of Vāta vyādhi, based on modern and ayurveda perspective;

Ativyavāya – Donald L Hilton and others in their research paper on pornography addiction: A neuroscience perspective, were of the opinion that compulsive sexuality can indeed be addictive. It concludes for the first time that a sexual compulsion can cause physical, anatomic change in the brain, the hallmark of brain addiction. A preliminary study showed frontal dysfunction specifically in patients unable to control their sexual behavior. The study used diffusion MRI to evaluate function of nerve transmission through white matter. It demonstrated abnormality in the superior frontal region, an area associated with compulsivity. Hormonal changes similar to overeating induced obesity were also observed.

Ati plavana, atiadva, ati vyāyāma, ati vichesta: Normal exercise has a good neurobiological impact. It increases the secretion of positive hormones and also helps in neurogenesis whereas over exercising can lead to an increased resting heart rate, a cause for increased cardiac output leading to hypertension, risk factor for stroke. Unexplained weight loss and decreased appetite is another factor. Further decreased of essential elements leads to neurological deficits as discussed before.

Further cortisol and stress hormones levels tend to increase with decrease in testosterone levels.

Emotions are intimately linked with organic life. They either result in an, “abnormal excitation of the nervous network, which induces changes in heart rate and secretions, or interrupts the normal relationship between the peripheral nervous system and brain.” Cerebral activity is focused on the source of emotions; voluntary muscles may become paralyzed and sensory perceptions may be altered including the feeling of physical pain. The idea of emotions involves

specific areas of brain and activation of these areas is associated with increase blood supply.

31 March 2016

Ch.Chi 28/ 15-19;

### **Etiopathogenesis of vaata vyaadhi -**

Most of the etiological factors explained here are responsible for kevala vāta prakopa mediated through dhātu kṣaya. Exceptions are less in the said group. But now days etiological factors causing āvarana or samsarga vāta prakopa are mostly found. This is because of increased standard of living. The so called neuro degenerative diseases like Parkinsonism Disease, Alzheimers Disease etc even now days considered as aftereffects of metabolic dysfunctions rather than under nutrition or overuse. Even diseases like Alzheimers dementia is conceptualised recently as type 3 Diabetes Mellitus.[20]

So a reassessment of etiological factors of contemporary importance is valid. Here an attempt is made to analyse the properties causing vāta vitiation with some modern explanations.

Rūkṣa, śītā, alpa, laghu anna, abhojana:

As any other organ, the brain is elaborated from substances present in the diet (sometimes exclusively, for vitamins, minerals, essential amino acids and essential fatty acids, including omega-3 polyunsaturated fatty acids). Most micronutrients (vitamins and trace elements) have been directly evaluated in the setting of cerebral functioning for e.g. vitamin B1 modulates cognitive performance

especially in elderly. Vitamin B9 preserves brain during its development and memory during ageing. Vitamin B6 is used in treating premenstrual depression. Vitamin B6 and B12, among others, are directly involved in the synthesis of some neurotransmitters. Vitamin B12 delays the onset of signs of dementia. Supplementation of Cobalamin improves cerebral and cognitive functions in the elderly. In the brain, the nerve endings contain the highest concentration of vitamin C in the human body (after the supra renal glands). Vitamin D (or certain of its analogues) could be of interest in the prevention of various aspects of neurodegenerative or neuro-immune diseases. Iron is necessary to ensure oxygenation and to produce energy in the cerebral parenchyma and for the synthesis of neurotransmitters and myelin. An unbalanced copper metabolism homeostasis (due to dietary deficiency) could be linked to Alzheimer's disease. Among many mechanisms manganese, copper and zinc participate in enzymatic mechanisms that protect against free radicals, toxic derivatives of oxygen. Indeed, nutrient composition and meal pattern can exert either immediate or long term effects beneficial or adverse.

From the above discussion it is observed that rūkṣa, alpa, laghu anna are apatarpankar hetu. Thus nutritional deficiency causes disorders of nervous system.

Similarly snigdha guna is essential to traverse the lipid soluble essential elements across the cell membrane. Rūkṣa guna in excess reduces the transfer of essential elements into the cells thus causing immediate or late effects.

Further diets that are rich in saturated fats and sugar decrease levels of Brain derived neurotrophic factor [BDNF]. BDNF is a neurotrophin considered generally beneficial for maintaining neuronal function and for promoting recovery after neurologic insult. Reduced BDNF leads to poorer neuronal performance. Results of a study have shown that rats fed on a diet high in saturated fats and refined sugars (similar in content to the "junk food" that has become popular in western society) for a period of 1 -2 months performed significantly worse on the spatial learning water maze test. Even more alarming is that the high fat diet



consumption exacerbated the effects of experimental brain injury. The effects of this high caloric diet seem to be related to elevated levels of oxidative stress and reduced synaptic plasticity which can be reversed by antioxidant treatment or exercise. High caloric intake also is perceived as a risk factor for Alzheimer's disease. Concept of atibhojana, snigdha etc. leads to āma utpatti, a cause for Vātavyādhī. Research results show that noninvasive approaches such as diet and exercise can have profound consequences for increasing resilience of the CNS to injuries and for maintaining cognitive abilities. Diet and exercise are 2 very important parts of lifestyle and daily routine each can influence the capability of the brain to fight disease and to react to challenges. Physical activity can benefit neuronal function and plasticity by enhancing synaptic plasticity and reducing oxidative stress. Physical exercise can have direct effects on the brain and spinal cord by supporting the maintenance of the synaptic structure, axonal elongation and neurogenesis in the adult brain whereas excessive exertion (ativyāyāma) is held to cause degenerative changes.

Stress is unpleasant, even when it is transient. A stressful situation—whether something environmental or psychological can trigger a cascade of stress hormones that produce well orchestrated physiological changes. Fight and flee are the 2 responses which the body is accustomed to. Repeated stress leads to hormonal and neuro-adaptive changes which may be the cause for damage. All krodha (fight response) and bhaya (flight response) described by Ācharyas explain the similar effects on the body.

Śītā guna- Prolonged exposure to the cold causes the body to slow blood circulation to the periphery. The reduced blood flow can intensify neuropathy symptoms and potentially cause further damage to already affected peripheral nerves.

Research is essential to rule out effects of śītā guna on cryoglobulinemia; a condition of cold antibody in blood which causes vasculitis and neuropathy as well.



Increase cryoglobulinemia increases viscosity leading to reduced blood flow thereby causing neuropathy.

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17 Feb 2016

My approach to Ch.Chi. 28...

VERSE 5-11 ; **vaata shaarira**

Modern anatomical or functional correlation of subtypes of vāta is attempted here for a rough and overall understanding for beginners. Prāna Vāyu is concerned with consciousness, arousal, heartbeat, vomiting, breathing, cough, hiccup etc. The modern functional analogue may be compared with brain stem and reticular formation which directly control cardiovascular / respiratory systems, pain sensitivity, alertness, awareness, and consciousness. Udāna is concerned with language, learning, mood, initiation, judgment, intellect, recall information etc. The prefrontal cortex, sub cortical areas and parts of limbic system along with association areas may be understood as functional areas of Udāna. Vyāna is concerned with control of skeletal muscle activities, control of hemodynamics, sweating etc. Post-lateral and dorso-medial hypothalamus - sympathetic stimulator, primary motor area, basal ganglia, extra pyramidal tract and autonomous nervous system are part and parcel of vyāna vāta. Samāna and Apāna can be considered together. Gastro Intestinal Tract based enteric nervous system (2nd brain), (brain- gut axis - more than 100 million neurons), celiac plexus, sacral plexus etc may be analogue for apāna and samāna.

The functioning of panch vāta prakār can be also understood by understanding the physiology of sensation. In its broadest definition, sensation is the conscious or subconscious awareness of changes in the external or internal environment. The nature of the sensation and the type of reaction generated vary according to the ultimate destination of nerve impulses that convey sensory information to the CNS. Sensory impulses that reach the spinal cord may serve as input for spinal reflexes, such as the stretch reflex, sensory impulses that reach the lower brain stem elicit more complex reflexes, such as changes in heart rate or breathing rate.

When sensory impulses reach the cerebral cortex, person become consciously aware of the sensory stimuli and can precisely locate and identify specific sensations such as touch, pain, hearing, or taste. Perception is the conscious awareness and interpretation of sensations and is primarily a function of the cerebral cortex. Person may have no perception of some sensory information because it never reaches the cerebral cortex. For example, certain sensory receptors constantly monitor the pressure of blood in blood vessels. Because the nerve impulses conveying blood pressure information propagate to the cardiovascular center in the medulla oblongata rather than to the cerebral cortex, blood pressure is not consciously perceived. Thus some functions may involve all the panch prakāra vāta and in some their permutation and combination.

#### Process of sensation

An appropriate stimulus must occur within the sensory receptor's receptive field, that is, the body region where stimulation activates the receptor and produces a response.

A sensory receptor transduces (converts) energy in a stimulus into a graded potential. Conversion of energy from one form to another i.e. transformation is the function of agni but the one which stimulates the agni is the samāna vāyu (agni samipasta and swedavaha (at the level of tvak) āshrayi vāta prakar). For example, odorant molecules in the air stimulate olfactory (smell) receptors in the nose, which transduces the molecules' chemical energy into electrical energy in the form of a graded potential.

When a graded potential in a sensory neuron reaches threshold, it triggers one or more nerve impulses, which then propagate toward the CNS. It explains the sarvasrotogata vyāna vāta action to take the nerve impulse towards the CNS. A particular region of the CNS receives and integrates the sensory nerve impulses. Conscious sensations or perceptions are integrated in the cerebral cortex.



Integration is the role of antahkarana but carried out by niyanta ca manasā i.e. vāta especially the prāna vāyu in this case.

A characteristic of most sensory receptors is adaptation, in which the generator potential or receptor potential decreases in amplitude during a maintained, constant stimulus. Because of adaptation, the perception of a sensation may fade or disappear even though the stimulus persists. For example, when you first step into a hot shower, the water may feel very hot, but soon the sensation decreases to one of comfortable warmth even though the stimulus (the high temperature of the water) does not change. This is the smriti kriya exhibited by the antahkaran but now with the help of udāna vāyu.

Many somatic motor neurons are regulated by the brain. When activated, somatic motor neurons convey motor output in the form of nerve impulses along their axons, which sequentially pass through the anterior gray horn and anterior root to enter the spinal nerve. From the spinal nerve, axons of somatic motor neurons extend to skeletal muscles of the body. This is again the function of vyāna. Thus afferent conduction of nerve impulse is the urdhwagati of vyāna, conduction from motor neurons to the skeletal muscle is the adhogati of vyāna and the autonomic nervous stimulation is the tiryaka gati of vyāna vāyu. This is the reason why Caraka in context of treatment of vāyu prakār has told “tridha vyānam tu yojayet” it explains vyāna has all the three gati which need to be regularize during the treatment.

The part of the body that responds to the motor nerve impulse, such as a muscle or gland, is the effector. Its action is called a reflex. If the effectors are skeletal muscle, the reflex is a somatic reflex. If the effectors are smooth muscle, cardiac muscle, or a gland, the reflex is an autonomic (visceral) reflex.

Depending on the resultant action function of vāta prakāra have been explained i.e. ṣṭhīvāna, kṣavathū, anna pravesha, udgār, nīswasa karma is seen that it is due to prāna vāyu.



Vākpravṛtti, prayatna, urjā, bala varna smṛiti are karma of udāna vāyu.

Anna vivechan, agni bala prada karma is due to samāna vāyu whereas ākuncan prasāran is due to vyāna vāyu and garbha, mūtra, purisa niskraman is due to apāna vāyu.

Thus the classification done is on the gross level of functioning. Similarly at cellular level too one can understand the existence of panch prakar vāta.

The selective permeability of the plasma membrane allows a living cell to maintain different concentrations of certain substances on either side of the plasma membrane. A concentration gradient is a difference in the concentration of a chemical from one place to another, such as from the inside to the outside of the plasma membrane. Many ions and molecules are more concentrated in either the cytosol or the extracellular fluid. For instance, oxygen molecules and sodium ions (Na) are more concentrated in the extracellular fluid than in the cytosol; the opposite is true of carbon dioxide molecules and potassium ions (K). The plasma membrane also creates a difference in the distribution of positively and negatively charged ions between the two sides of the plasma membrane. Typically, the inner surface of the plasma membrane is more negatively charged and the outer surface is more positively charged. A difference in electrical charges between two regions constitutes an electrical gradient. Because it occurs across the plasma membrane, this charge difference is termed the membrane potential. In many cases a substance will move across a plasma membrane down its concentration gradient. That is to say, a substance will move "downhill," from where it is more concentrated to where it is less concentrated, to reach equilibrium. Similarly, a positively charged substance will tend to move toward a negatively charged area, and a negatively charged substance will tend to move toward a positively charged area. The combined influence of the concentration gradient and the electrical gradient on movement of a particular ion is referred to as its electrochemical gradient.

Transport of materials across the plasma membrane is essential to the life of a cell. (āyu is one of the paryāya of vāyu). Certain substances must move into the cell to support metabolic reactions (pravesakrita karma of prāna vāyu). Other substances that have been produced by the cell for export or as cellular waste product (niskramana karma of apāna vāyu) must move out of the cell.

The concentration gradient which is maintained is essential for cellular activity. Resting membrane potential and active membrane potential are maintained at specific levels. For e.g. Charges of -90 mv is the resting charge which reaches to +35 mv when depolarized in cardiac cell thus this knowledge of potential gradient is due to budhi dharan karma of prāna which cause the pumping of Na/K pump to activate. Thus knowledge of concentration gradient is karma of prāna vāyu. Further prāna means prinana ādāna karma i.e. helping entry/ facilitation of such ions, essential requirements within cell which will do prinan /poshan is also due to prāna. Thus process that initiates endocytosis is prāna vāyu.

Substances generally move across cellular membranes via transport processes that can be classified as passive or active, depending on whether they require cellular energy. In passive processes, a substance moves down its concentration or electrical gradient to cross the membrane using only its own kinetic energy. The continuous movement resembles the cala guna, a common quality of all the types of vāta. Modern describes it as the Brownian movement of the ions. Kinetic energy is intrinsic to the particles that are moving. There is no input of energy from the cell. An example is simple diffusion.

In active processes, cellular energy is used to drive the substance “uphill” against its concentration or electrical gradient. The cellular energy used is usually in the form of ATP. It explains the prayatna karma of udāna vāyu which is responsible for the activity. An example is active transport. Active transport is considered an active process because energy is required for carrier proteins to move solutes across the membrane against a concentration gradient. Two sources of cellular energy can be used to drive active transport: (1) Energy obtained from hydrolysis

of adenosine triphosphate (ATP) is the source in primary active transport; (2) energy stored in an ionic concentration gradient is the source in secondary active transport. Like carrier-mediated facilitated diffusion, active transport processes exhibit a transport.

Many of the infolding of the inner membrane form shelves on which oxidative enzymes are attached. In addition, the inner cavity of the mitochondrion is filled with a matrix that contains large quantities of dissolved enzymes that are necessary for extracting energy from nutrients. These enzymes operate in association with the oxidative enzymes on the shelves to cause oxidation of the nutrients, thereby forming carbon dioxide and water and at the same time releasing energy. The liberated energy is used to synthesize a "high-energy" substance called adenosine triphosphate (ATP). ATP is then transported out of the mitochondrion, and it diffuses throughout the cell to release its own energy wherever it is needed for performing cellular functions. Thus the phenomenon which triggers the oxidative process is the samāna vāyu which stimulates the oxidation i.e. role of agni.

The intracellular movement of proteins, ATP transfer, and vesicle transportation can be understood as the vyāpan/ vyuhan karma of vyāna vāyu.

The end metabolites formed within the cell are removed through the process of exocytosis. The process is initiated by apāna vāyu which helps in excretion, mokshan, munchan karma at the level of cel

15 Feb 2016

### **My approach to vaatasya karma ;**

The normal electrical conduction in the heart allows the impulse that is generated by the sinoatrial node (SA node) of the heart to be propagated to, and stimulate, the cardiac muscle (myocardium). The myocardium contracts after stimulation. It

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is the ordered, rhythmic stimulation of the myocardium during the cardiac cycle that allows efficient contraction of the heart, thereby allowing blood to be pumped throughout the body. Signals arising in the SA node (located in the right atrium) stimulate the atria to contract and travel to the AV node, which is located in the interatrial septum. After a delay, the stimulus diverges and is conducted through the left and right bundle of His to the respective Purkinje fibers for each side of the heart, as well as to the endocardium at the apex of the heart, then finally to the ventricular epicardium.

On the microscopic level, the wave of depolarization propagates to adjacent cells via gap junctions located on the intercalated disk. The heart is a functional syncytium (not to be confused with a true "syncytium" in which all the cells are fused together, sharing the same plasma membrane as in skeletal muscle). In a functional syncytium, electrical impulses propagate freely between cells in every direction, so that the myocardium functions as a single contractile unit. This is the avyahata gati of vata which is necessary for the rapid, synchronous depolarization of the myocardium. Conduction from SA to AV to bundles and Purkinje fiber is the aparityakta swa marga of vata. This rhythmical and conductive system of the heart is susceptible to damage by heart disease, especially by ischemia of the heart tissues resulting from poor coronary blood flow. The result is often a bizarre heart rhythm or abnormal sequence of contraction of the heart chambers, and the pumping effectiveness of the heart often is affected severely, even to the extent of causing death. This explains the vyahat gati of vata which is the cause of death

The circulatory system is the main method for blood transportation within body. This system is a complex highway of vessels, and its main purpose is to move blood and nutrients throughout body. The circulatory system is also responsible for exchanging gases and removing waste products from body. Unlike an open circulatory system, a closed circulatory system is more structured and controlled. The blood of a closed system always flows inside vessels. These vessels make up



the plumbing circuit of the body and can be found throughout the entire body. This plumbing circuit can be broken down into three different types of vessels, or tubes that transport blood throughout the body: arteries, capillaries and veins. Thus a continuous flow of blood from Left ventricles to the aorta to arteries all over the body than to arterioles into capillaries into venules into veins and back to the right atrium than right ventricle via pulmonary artery to the lungs and via pulmonary veins to the left atrium and back to left ventricle. This is how blood is propagated from heart to the periphery and back to the heart.

Vyanena rasadhatuhi vikshepa uchita karmana yugpat sarvato ajashram dehe vikshepyate sada || Ca. Ci. 15/36

This function of vata is swa sthanastha which helps to maintain the homeostasis or swasthya but when avarodh to this gati takes place may be due to any reason the swa margasthita vata gets vimargagata as explained in samprapti of shoth.

Bahya sira prapya yada kapha asruka pittani sandushayati vayu || Ca. Ci. 12/8

Various edema are either due to excessive secretion (apana vayu) or reduced absorption (pran vayu) as understood in samprapti of udara. Disturbed concentration of solutes and solvents causes changes in pressure (vyan vayu) either intravascular or extra vascular. The electrolyte balance is brought about by sweda dosha ambu srotas sthayi vayu i.e. samana vayu.

Prakruti sthita vata is the one which is akshina vridha. Normal pulse rate ranges from 60-80/min. Excessive pulse rate explains the repeated contraction of heart one of the cause being excessive aakunchan prasaran karma of vyan vridhi whereas one of the cause of bradycardia may be kshina vyan vayu.

Increased peristalsis is the cause for increased frequency of stools one of the reason being vridha apana vata whereas reduced peristalsis causes constipation one reason being kshina apana vata.

Excessive stimulation of agni (atyagni) causes increased appetite one reason being vridha samana vata whereas agnimandhya, grahani etc may be caused by decrease stimulation of agni by samana vata.

Prayatna, urja are functions of udan vayu. Excessive excitation of cell due to excess action potential explains the vridha udan vayu whereas inhibition of cell activity due to reduced action potential is due to kshina udan vayu.

Reduced respiratory rate due to depressed respiratory centre explains kshin prana vayu whereas vridha pran vayu may be one of the cause or increased ventilation.

The vata dosha on basis of its functions is classified into five types. They reside in the sharir at the level of sharir parmanu (cell) and also at gross level. The five prakara work together in a synchronized manner for the normal functioning of the sharir (vayu tantrayantra dhara). In this context upamana pramana of people with different profession like malakara, kumbakar stay together under one roof is given.

The functioning of panch vata prakar can be understood by understanding the physiology of sensation. In its broadest definition, sensation is the conscious or subconscious awareness of changes in the external or internal environment. The nature of the sensation and the type of reaction generated vary according to the ultimate destination of nerve impulses that convey sensory information to the CNS. Sensory impulses that reach the spinal cord may serve as input for spinal reflexes, such as the stretch reflex, sensory impulses that reach the lower brain stem elicit more complex reflexes, such as changes in heart rate or breathing rate. When sensory impulses reach the cerebral cortex, person become consciously aware of the sensory stimuli and can precisely locate and identify specific sensations such as touch, pain, hearing, or taste. Perception is the conscious awareness and interpretation of sensations and is primarily a function of the cerebral cortex. Person may have no perception of some sensory information

because it never reaches the cerebral cortex. For example, certain sensory receptors constantly monitor the pressure of blood in blood vessels. Because the nerve impulses conveying blood pressure information propagate to the cardiovascular center in the medulla oblongata rather than to the cerebral cortex, blood pressure is not consciously perceived. Thus some functions may involve on the panch prakara vata and in some their permutation and combination.

#### Process of sensation

An appropriate stimulus must occur within the sensory receptor's receptive field, that is, the body region where stimulation activates the receptor and produces a response.

A sensory receptor transduces (converts) energy in a stimulus into a graded potential. Conversion of energy from one form to another i.e. transformation is the function of agni but the one which stimulates the agni is the samana vayu (agni samipasta and sweda vaha (at the level of tvak) ashrayi vata prakar). For example, odorant molecules in the air stimulate olfactory (smell) receptors in the nose, which transduces the molecules' chemical energy into electrical energy in the form of a graded potential.

When a graded potential in a sensory neuron reaches threshold, it triggers one or more nerve impulses, which then propagate toward the CNS. It explains the sarvasrotogata vyan vata action to take the nerve impulse towards the CNS. A particular region of the CNS receives and integrates the sensory nerve impulses. Conscious sensations or perceptions are integrated in the cerebral cortex. Integration is the role of antakarana but carried out by niyanta cha manasa i.e. vata especially the pran vayu in this case.

A characteristic of most sensory receptors is adaptation, in which the generator potential or receptor potential decreases in amplitude during a maintained, constant stimulus. Because of adaptation, the perception of a sensation may fade or disappear even though the stimulus persists. For example, when you first step



into a hot shower, the water may feel very hot, but soon the sensation decreases to one of comfortable warmth even though the stimulus (the high temperature of the water) does not change. This is the smriti kriya exhibited by the antakaran but now with the help of udan vayu.

Many somatic motor neurons are regulated by the brain. When activated, somatic motor neurons convey motor output in the form of nerve impulses along their axons, which sequentially pass through the anterior gray horn and anterior root to enter the spinal nerve. From the spinal nerve, axons of somatic motor neurons extend to skeletal muscles of the body. This is again the function of vyan. Thus afferent conduction of nerve impulse is the urdhwagati of vyan, conduction from motor neurons to the skeletal muscle is the adhogati of vyan and the autonomic nervous stimulation is the tiryaka gati of vyan vayu. This is the reason why Caraka in context of treatment of vayu prakar has told “tridha vyanam tu yojayet” it explains vyan has all the three gati which need to be regularize during the treatment.

The part of the body that responds to the motor nerve impulse, such as a muscle or gland, is the effector. Its action is called a reflex. If the effectors are skeletal muscle, the reflex is a somatic reflex. If the effectors are smooth muscle, cardiac muscle, or a gland, the reflex is an autonomic (visceral) reflex.

Depending on the resultant action function of vata prakar have been explained i.e. ksthivan, kshavathu, anna pravesha etc karma is seen that it is due to pran vayu.

Vata pravrutti, prayatna, urja, bala varna smriti etc are karma of udan vayu.

Anna vivechan, etc karma is due to samana vayu whereas aakunchan prasara is due to vyan vayu and garbha nischikraman etc is due to apana vayu.

Thus the classification done is on the gross level of functioning. Similarly at cellular level too we can understand the existence of panch prakar vata.



The selective permeability of the plasma membrane allows a living cell to maintain different concentrations of certain substances on either side of the plasma membrane. A concentration gradient is a difference in the concentration of a chemical from one place to another, such as from the inside to the outside of the plasma membrane. Many ions and molecules are more concentrated in either the cytosol or the extracellular fluid. For instance, oxygen molecules and sodium ions (Na) are more concentrated in the extracellular fluid than in the cytosol; the opposite is true of carbon dioxide molecules and potassium ions (K). The plasma membrane also creates a difference in the distribution of positively and negatively charged ions between the two sides of the plasma membrane. Typically, the inner surface of the plasma membrane is more negatively charged and the outer surface is more positively charged. A difference in electrical charges between two regions constitutes an electrical gradient. Because it occurs across the plasma membrane, this charge difference is termed the membrane potential. In many cases a substance will move across a plasma membrane down its concentration gradient. That is to say, a substance will move “downhill,” from where it is more concentrated to where it is less concentrated, to reach equilibrium. Similarly, a positively charged substance will tend to move toward a negatively charged area, and a negatively charged substance will tend to move toward a positively charged area. The combined influence of the concentration gradient and the electrical gradient on movement of a particular ion is referred to as its electrochemical gradient.

Transport of materials across the plasma membrane is essential to the life of a cell. (ayu is one of the paryaya of vayu). Certain substances must move into the cell to support metabolic reactions (praveshakruta karma of pram vayu). Other substances that have been produced by the cell for export or as cellular waste product (niskramana karma of apana vayu) must move out of the cell.

Substances generally move across cellular membranes via transport processes that can be classified as passive or active, depending on whether they require

cellular energy. In passive processes, a substance moves down its concentration or electrical gradient to cross the membrane using only its own kinetic energy. The continuous movement resembles the chala guna, a common quality of all the types of vata. Modern describes it as the Brownian Movement of the ions. Kinetic energy is intrinsic to the particles that are moving. There is no input of energy from the cell. An example is simple diffusion.

In active processes, cellular energy is used to drive the substance "uphill" against its concentration or electrical gradient. The cellular energy used is usually in the form of ATP. It explains the prayatna karma of udan vayu which is responsible for the activity. An example is active transport. Active transport is considered an active process because energy is required for carrier proteins to move solutes across the membrane against a concentration gradient. Two sources of cellular energy can be used to drive active transport: (1) Energy obtained from hydrolysis of adenosine triphosphate (ATP) is the source in primary active transport; (2) energy stored in an ionic concentration gradient is the source in secondary active transport. Like carrier-mediated facilitated diffusion, active transport processes exhibit a transport.

The concentration gradient which is maintained is essential for cellular activity. Resting membrane potential and active membrane potential are maintained at specific levels. For e.g. Charges of -90 mv is the resting charge which reaches to +35 mv when depolarized in cardiac cell thus this knowledge of potential gradient is due to budhi dharan karma of pran which cause the pumping of Na/K pump to activate. Thus knowledge of concentration gradient is karma of pran vayu. Further pran means prinana aadan karma i.e. helping entry/ facilitation of such ions, essential requirements within cell which will do prinan /poshan is also due to pran. Thus process that initiates endocytosis is pran vayu.

Many in folding of the inner membrane form shelves onto which oxidative enzymes are attached. In addition, the inner cavity of the mitochondrion is filled with a matrix that contains large quantities of dissolved enzymes that are

necessary for extracting energy from nutrients. These enzymes operate in association with the oxidative enzymes on the shelves to cause oxidation of the nutrients, thereby forming carbon dioxide and water and at the same time releasing energy. The liberated energy is used to synthesize a "high-energy" substance called adenosine triphosphate (ATP). ATP is then transported out of the mitochondrion, and it diffuses throughout the cell to release its own energy wherever it is needed for performing cellular functions. Thus the phenomenon which triggers the oxidative process is the samana vayu which stimulates the oxidation i.e. role of agni.

The intracellular movement of proteins, ATP transfer, and vesicle transportation can be understood as the vyapan/ vyuhan karma of vyan vayu.

The end metabolites formed within the cell are removed through the process of exocytosis. The process is initiated by apana vayu which helps in excretion, mokshan , munchan karma at the level of cell.

**Ardita - अर्दित - चरकोक्त ( च.चि.28 ) - the neuroscience ;**

Ardita may be understood as many clinical conditions. Normally it is diagnosed as Bell's palsy. The facial paralysis may be either of Upper Motor Neuron (UMN) or Lower Motor Neuron (LMN) origin. The etiological factors explained in Ashtanga Hridaya like bearing weight over head, over exertion activities to temporomandibular joint etc may lead to local causes to form LMN pathology. Here as Ardita is explained as vegavān disease clinical conditions like TIA and RIND etc also may be understood in terms of Ardita. Further Hemifacial spasm, synkynesis etc has an after effect of poor managed or unresolved facial palsy is also understood as ardita.

Caraka from shlok no. 38 onwards has classified the disease on basis of presenting symptoms. Since even in modern classification one may find neurological deficits become difficult to classify on basis of disease because for example facial presentation may be due to Trigeminal nerve, facial nerve, infective as in herpes, tumour like brain lesion, infarct or hemorrhagic in origin. Irrelevant of aetiological factors one needs give importance to the symptoms.

In context of facial palsy may be / may not be associated with involvement of other parts of the body. A complete interruption of the facial nerve at the stylo-mastoid foramen paralyzes all muscles of facial expression. The corner of mouth droops, the creases and skin-fold are effaced, the forehead is unfurrowed and the eyelids will not closed [stabdham nētram]. Upon attempted closure of the lids, the eye on the paralyzed side rolls upward (Bells phenomenon), food collects between the teeth and lips and saliva may dribble from the corner of the mouth [vakram vrajatyāsyē bhōjanam]. If the nerve to the stapedius is interrupted, there is hyperacusis (sensitivity to loud sounds). Lesions in the internal auditory meatus may affect the adjacent auditory and vestibular nerves, causing deafness, tinnitus or dizziness (bādhyētē śravaṇau). Chakrapāni has explained it to be prabhav but



today after complete anatomical study above explanation for hearing deficit may be proved.

If the peripheral facial paralysis has existed for some time and recovery of motor function is incomplete, a continuous diffuse contraction of facial muscles may appear.

Further other than Bells palsy facial palsy may be seen unilaterally or bilaterally in Lymes Disease. The Ramsay Hunt Syndrome caused by reactivation of Herpes zoster in the geniculate ganglion. Facial palsy that is often bilateral occurs in sarcoidosis and in Guillain Barre Syndrome, Leprosy, Diabetes mellitus, connective tissue diseases including Sjogren's syndrome and Amyloidosis. The rare Melkersson Rosenthal Syndrome consists of recurrent facial paralysis and tumors of temporal bone.

In supranuclear lesion there may be a dissociation of emotional and voluntary facial movements and often some degree of paralysis of arm or leg or aphasia.

Further in corticobulbar involvement, weakness is usually observed only in the lower face and tongue; extra ocular, upper facial pharyngeal and few muscles are almost always spared. With bilateral corticobulbar lesions, pseudobulbar palsy often develops: dysarthria, dysphagia, dysphonia and emotional lability accompany bilateral facial weakness and a brisk jaw jerk. A "pure motor" hemiparesis of the face, arm or leg is often due to a small, discrete lesion in the posterior limb of the internal capsule, cerebral peduncle or upper pons. Some brainstem lesion produces "crossed paralysis" consisting of ipsilateral cranial nerve signs and contralateral hemiparesis.

From above discussion one can understand various presentation of facial palsy which may / may not be associated with involvement of other parts of the body. Misdirection of food, resulting in nasal regurgitation and laryngeal and pulmonary aspiration during swallowing is characteristic of oropharyngeal dysphagia [bhōjanam vakranāsikam].

Vakram vrajatyāsyē, Chakrapāni comments, nā samam mukhena khādati kintu vakra ekadeshena explains the weakness of oral muscle due to which patient is unable to chew and swallow equally from both the sides.

Dinā jihya samutkshipta kalā sajjati ca āsya vāka explains the language disturbances (aphasia) seen in such patients. Aphasia should be diagnosed only when there are deficits in the formal aspects of language such as naming, word choice, comprehension, spelling and syntax. The neural substrate of language is composed of a distributed network centered in the perisylvian region of the left hemisphere. The posterior pole located at temporoparietal junction and includes region known as Wernicke's area and the anterior pole of language network is known as Broca's area. Both this area are interconnected with each other with additional perisylvian, temporal, prefrontal and posterior parietal regions making up a neural network sub serving the various aspects of language function.

Anomia – deficit of naming

Paraphasia – Name the object with wrong word or fail to come up with appropriate word, may provide a circum-locutious description of the object.

Semantic paraphasia – If patient offers an incorrect but legitimate word (pen for pencil) the naming error is semantic paraphasia.

Phoenemic paraphasia – word approximates correct answer but is phonetically inaccurate (plentil for pencil).

Spontaneous speech [samutshipta atitvarita] is described as “fluent” if it maintains appropriate output volume, phrase length and melody or as “non fluent” if it is sparse, halting and overage utterance length below four words [kaleti avyaktā]

Alexia describes an inability to either read aloud or comprehend single words and simple sentences.

7 August 2014

Facial nerve paralysis ; Ardita-; Confirm whether upper motor or lower motor neuron lesion is present ; Test eyebrow elevation , forehead wrinkling , eye closure , smiling and cheek puff. Look in particular for differences in the lower versus upper facial muscles; weakness of lower two thirds of face with preservation of the upper third suggests an upper motor neuron lesion, whereas weakness of an entire side suggests a lower motor neuron lesion ( Bell' s palsy ).

Viral Infections (HSV) are common cause of LMN lesion and HTN is common cause of UMN lesion.. Manage BP by beta blockers. prednisolone for LMN lesion , no need of anti viral drugs.. ekangaveera rasa , mahayogaraaja guggul , vishatinduka vati , kvaatha of dashamoola , ashwagandha, shatavari, bala , kapikachchhu ,maasha chopachini and amrita. And snehan , svedan , nasya , moordha tail (as told by acharya charak ; ch.chi 28/99-100)

Majja kshaya and its clinical perspective ; Mridu , laghu, sookshma, shlakshna , shabda are guna of aakaasha so their karma are maardava, saushirya, laaghava...due to maardavataa its apratighaatatva...( due to ) aakaasha baahulyam dravyasya pruthivyaadi bhootaantarah alpatvena bhoori vyakta aakaashatvena cha gyeyam chakrapani on ch.su. 26/11..

In majjaa kshaya shiryanta eva cha ashttheeni durbalaani laghooni cha..

In majjaa kshaya prithivyadi bhootaantarah alpatven bhavati.. so aakaasha guna is increased..

Osteoporosis and osteopenia are best example.. kamdudha/pravaala panchaamrita ,pancha tikta ghrit guggul, ashvagandhaa, laksha,arjuna,padmyakaashtha, amrita, aamalaki,mustaa like drugs are indicated...

2 June 2013

Ardhe tasminmukhardhe vaa kevale syaattadarditam ch.chi28/42.. Facial paralysis or faciobrachiocrural paralysis ( facial paralysis with ipsilateral hemiplegia)...

## Pakshaghat

Most beautiful clinical approach in few lines ; हृत्वेकं मारुतः पक्षं दक्षिणं वाममेव वा ।  
कुर्यात् चेष्टा निवृत्तिं हि रुजं वाक्स्तम्भमेव च , गृहीत्वा अर्धं शरीरस्य सिराः स्नायूः  
विशोष्य च। पादं संकोचयति एकं हस्तं वा तोदशूलकृत् , एकांगरोगं तं विद्यात् सर्वांगं  
सर्वदेहजम् ।।च.चि.28/53-55

In these verses Ācharya Caraka explains three paralytic disorders namely monoplegia (ekāngarōga), hemiplegia (pakshavadha) and quadriplegia (sarvāngarōga). In Ashtanga Hridaya ekāngarōga and pakshavadha are explained as synonyms. According to Caraka ekāngarōga is monoplegia which is associated with toda and shoola which seems to be peripheral radiculopathy with motor deficit or focal brain lesions. In pakshavadha paralysis, aphasia and sensory symptoms may co exist. Paralysis is loss of muscle function for one or more muscles. It may be accompanied by a sensory loss.

### Causes

- Stroke – a) Hemorrhagic b) Ischemic
- Trauma with nerve injury
- Cerebral palsy
- Poliomyelitis
- Cerebral palsy
- Peripheral neuropathy



- Parkinson's disease
- Spina bifida
- Multiple sclerosis
- Amyotrophic Lateral Sclerosis
- Botulism
- Guillain Barre Syndrome

Hatvaikaṁ: – explains loss of voluntary movement either in one part /or whole body. It usually refers to the limbs either mono i.e. one leg or one arm [ekāṅgarōga] or para i.e. both legs, hemi i.e. one arm and one leg on either side of the body [dakṣiṇaṁ vāmam ēva vā] and quadri i.e. all four limbs (sarvāṅga rōga).

Cēṣṭā nivṛttiṁ is seen due to loss of muscle power and tone.

Rujā /pain are due to reduced venous return leading to increase lactic acid within the muscle causing the pain. Thus physiotherapy helps to reduce pain.

Sirāḥ Snāyūrviśōṣya – explains atrophy i.e. partial or complete wasting of a part of the body. Poor nourishment, poor circulation, loss of nerve supply and disuse or lack of exercise is the cause.

Pādaṁ saṅkōcayatyēkaṁ hastaṁ vā: Damage to the pyramidal tract and its accompanying para pyramidal (cortico-reticulospinal) fibers give rise to UMN syndrome including positive and negative features. Impaired ability of damaged motor neurons to regulate descending pathways gives rise to disordered spinal reflexes, increased excitability of muscle spindles and decreased synaptic inhibition. This results in increased muscle tone of symptomatic muscle. The increased muscle tone may further lead to contractures.

Stroke symptoms typically start suddenly, over seconds to minute and in most cases do not progress further. The more extensive area of brain affected, more functions are likely to be lost.

Symptoms as per affected area:

A) CNS pathways – Spinothalamic tract, corticospinal tract and dorsal column

- Hemiplegia and muscle weakness of face
- Numbness
- Reduction in sensory or vibratory sensation
- Initial flaccidity (reduced muscle tone), replaced by spasticity (increased muscle tone), excessive reflexes and obligatory synergy.

B) If Brainstem which gives rise to cranial nerve gets involved, than

- altered smell, taste, hearing or vision
- drooping of eyelid (ptosis) and weakness of ocular muscle
- decreased reflexes: gag, swallow, pupil reactivity to light
- decreased sensation and muscle weakness of the face
- balance problems and nystagmus
- altered breathing and heart rate
- weakness in sternocleidomastoid muscle with inability to turn head on one side
- weakness in tongue

C) Cerebral cortex

- aphasia

- dysarthria
- apraxia
- visual field defect
- memory deficit
- disorganized thinking, confusion, hypersexual gestures (frontal lobe)

#### D) Cerebellum

- altered walking gait
- altered movement coordination
- vertigo and or disequilibrium..

2 June 2013

Pakshaaghaato angasanshoshah ( muscular atrophy) pangutvam(paraplegia or paraparesis) khudvaatataa( chakrapani: gulphvaatataa- foot drop). Ch.ch.28/73

25 August 2012

Ardhe tasminmukhardhe va kevale syattadarditam ch.chi 28/42 chakrapani:keval iti ardhe ev n kritsne.nanu yada deh ardha vyapitvamarditasya tada arditen ardhangasya ko bhedah? broomah:ardito vegitaya n sarv kalam bhavati,ardhangastu sarv kalam vyapya bhavati;uktam hi -svasthah syadarditadinam muhurvegagame gate iti. kivam yathokt vishisht lakshanoarditah;ardhange tu naitani sarvani bhavanti. in modern health science;facial paralysis,faciobrachiocrural paralysis. supranucleus and infranucleus types of facial paralysis.contralateral and ipsilateral faciobrachiocrural paralysis. e.g. Rt facial paralysis with Lt hemiplegia or Rt facial paralysis with Rt hemiplegia. perfect clinical observation of acharya charak and scientific interpretation by acharya chakrapani. with regards.

20 March 2015

**Dandaakshepaka** ; paani paadam cha sanshoshya sirah sasnaayu kandaraah. Paani paada shirah prishtha shronih stabhnaati maarutah ( ch.chi 28/51).. dystonia is a disorder characterised by sustained or repetitive involuntary muscle contractions frequently associated with twisting or repetitive movements and abnormal postures. Primary , focal , secondary and dystonia plus syndromes are present.. focal dystonias include blepharospasm , oromandibular dystonia , spasmodic dystonia , cervical dystonia , and limb dystonias.. snehan svedan vaataghna and maansa balya drugs..

Ataxic disorders ; manifest gait impairment , unclear speech , visual blurring due to nystagmus , hand incoordination , and tremors with movement. These result from the involvement of cerebellum and its afferent and efferent pathways.. progressive and symmetric ataxia can be classified as acute, sub acute, and chronic . Symmetric gait ataxia suggests an inherited ataxia, a metabolic disorders , or a chronic infection. Hypothyroidism must always be considered as a readily treatable and reversible form of gait ataxia.. muhuraakshipati kruddho gaatraani aakshepako anilah.. ( ch.chi.28/50) .. snehana , svedana , nasya or shirodhara , vaataghna and balya...cervical dystonia ; dystonic contractions of neck muscles causing the head to deviate to one side ( torticollis) , in a forward direction (anterosclerosis) , or in a backward direction ( retrocollis) .muscle contractions can be painful , and associated with a secondary cervical radiculopathy.. it can be correlated with manyastambha or antaraayaama but dantaanaam danshanam laalaa are not found in cervical dystonia , eventhough we can consider both as similar disease process and similar line of treatment. Physiotherapy should be encouraged in all types of neuromuscular diseases . swimming or walking or exercise in water (hydrotherapy ) is also best option , should be recommended..



23 March 2016

My observation ; यदि गृध्रसि का रुग्ण , स्फिक् पूर्व पृष्ठ कटि... वेदना युक्त है, compression is at L5-S1 level.. यदि पृष्ठ/ कटि पूर्व ... है , compression is at L3-4-5 level.. Similar diagnosis but with variable features.. depends on उद्भव स्थान.If knee jerk is diminished or absent indicates that the compression is at L3-4 levels , absent/diminished ankle jerk in S1-2 levels compression , and absent or diminished plantar reflexes show the compression is at L4 level.. By observing deep tendon reflexes in गृध्रसी रुग्ण , we can understand the exact levels of compression..I feel , the expansion of knowledge is need of hours !

20 Feb 2016

. GINGER

Ginger (*Zingiber officinale*) is a member of the Zingiberaceae family and is consumed widely not only as a spice but also as a medicinal agent (see also Chapter 7 on ginger). Other members of the family include turmeric and cardamom. Ginger's cultivation appears to have begun in South Asia and has now spread to various parts of the world. It is sometimes called "root ginger" to distinguish it from other products that share the name. The principal constituents of ginger include [6]-gingerol, [6]-paradol, [6]-shogaol (dehydration gingerols), and zingerone. Several studies have investigated ginger's antioxidant properties (Chrubasik, Pittler, and Roufogalis 2005). Gingerol has also been shown to decrease intracellular ROS formation in human keratinocyte cells (Kim et al. 2007), inhibit angiogenesis in human ECs, and limit nitrogen oxide synthase expression

and epidermal growth factor-induced cell transformation and AP-1 transcriptional complexes in JB6 cells (Bode et al. 2001; Ippoushi et al. 2003; Davies et al. 2005; Kim et al. 2005).

Feeding NIN/Wistar rats a diet containing up to 0.5-5% ginger for 1 month significantly increased ( $p < .05$ ) several liver antioxidant enzymes, including superoxide dismutase (76–141%), catalase (37–94%), and GPx (11–30%; Kota, Krishna, and Polasa 2008). Lipid and protein oxidation was inhibited in rats consuming ginger, as evidenced by significant decreases ( $p < .05$ ) in liver and kidney levels of MDA (35-59% and 27-59%, respectively) and carbonyl levels (23-36%), compared to controls (Kota, Krishna, and Polasa 2008). Ippoushi et al. (2007) found that AIN-76 basal diets with 2% ginger decreased TBARS by 29% ( $p < .05$ ) and suppressed 8-hydroxy-2'-deoxyguanosine (8-OHdG, a product of oxidative DNA damage) levels in Wistar rats. TBARS was also significantly decreased ( $p < .001$ ) in Wistar rats fed with diets supplemented with 1% ginger following exposure to lindane, a pesticide that is a global pollutant, (Ahmed et al. 2008).

Various animal models have been used to examine the role of ginger in cancer prevention. For example, Ihlaseh et al. (2006) exposed male Wistar rats to N-butyl-N-(4-hydroxybutyl)-nitrosamine (BNN) and uracil salt to induce tumors resembling human low-grade papillary urothelial neoplasia. Rats fed with a basal diet supplemented with 1% ginger extract for 26 weeks had significantly fewer urothelial lesions compared to the controls or those fed with the diet with 0.5% ginger ( $p = .013$ ; Ihlaseh et al. 2006). However, ginger does not appear effective in all cases, as evidenced by the lack of protection against proliferative lesions in the bladders of Swiss mice fed with a 1% or 2% extract and exposed to BNN/N-methyl-N-nitrosourea (Bidinotto et al. 2006).

Induction of phase I and II activities may partially account for ginger's anticarcinogenic actions. Banerjee et al. (1994) found that providing 10- $\mu$ L ginger oil daily for 2 weeks to Swiss mice increased aryl hydrocarbon hydroxylase activity about 25% ( $p < .05$ ) and increased GST by 60% ( $p < .01$ ). No significant increase in

GST induction was observed in Swiss mice fed with 160 mg ginger/gram diet (Aruna and Sivaramakrishnan 1990).

Inflammation is a significant risk factor for cancer, including prostate cancer. Mitogen-activated protein kinase phosphatase-5 (MKP5) is implicated as a proinflammatory inhibitor in innate and adaptive immune response in vivo (Zhang et al. 2004). Providing [6]-gingerol upregulated MKP5 expression in normal prostate epithelial cells treated with 50  $\mu$ M gingerol; likewise, it upregulated MKP5 expression in human prostate cancer cell lines (DU145, PC-3, LNCaP and LAPC-4; Nonn, Duong, and Peehl 2007). Ginger extracts, more so than their individual components, have been shown to inhibit lipopolysaccharide-induced prostaglandin E2 (PGE2) production to an extent similar to that of indomethacin, a nonsteroidal anti-inflammatory drug. Subfractions of ginger extract decreased LPS-induced COX-2 mRNA expression levels, although apparently not through the nuclear factor  $\kappa$ B (NF- $\kappa$ B) or activating protein 1 (AP-1) transcription factor pathways, because the ginger extracts did not inhibit TNF- $\alpha$  production (Lantz et al. 2007). [6]-paradol, another active compound in ginger, is reported to induce apoptosis in human promyelocytic leukemia cells, JB6 cells, an oral squamous carcinoma cell line, and Jurkat human T-cell leukemia cells in a dosedependent manner (Huang, Ma, and Dong 1996; Lee and Surh 1998; Keum et al. 2002; Miyoshi et al. 2003). It is unclear whether [6]-paradol has molecular targets similar to [6]-gingerol.

Ginger also appears to have antitumorigenic properties. Several cell lines have been examined for their sensitivity to ginger. For example, alcoholic extracts of ginger inhibited tumor cell growth for Dalton's lymphocytic ascites tumor cells and human lymphocytes at concentrations of 0.2-1 mg/mL in vitro (Unnikrishnan and Kuttan 1988). In a study of cytotoxic activities of several compounds in ginger against four tumor cell lines (A549, human lung cancer; SK-OV-3, human ovarian cancer; SK-MEL-2, human skin cancer; and HCT-15, human colon cancer), [6]-shogaol was the most potent (ED50: 1.05–1.76  $\mu$ g/mL), and [4]-, [6]-, [8]-, and



[10]-gingerol displayed moderate cytotoxicity (ED<sub>50</sub>: 4.92-30.05; Kim et al. 2008). Adding [6]-gingerol (25  $\mu$ M) has been reported to inhibit proliferation in rat ascites hepatoma cells AH109A and increase apoptosis at higher concentrations (50  $\mu$ M; Yagihashi, Miura, and Yagasaki 2008). Likewise, adding [6]-shogaol (60  $\mu$ M) to COLO295 cells has been reported to increase the expression of GADD153, a gene that promotes apoptosis (Chen et al. 2007). [6]-shogaol (>50  $\mu$ M) also provokes DNA damage and apoptosis through an oxidative stress-mediated caspase-dependent pathway (Chen et al. 2007). Similarly, incubation of HEP-2 cells with ginger (250  $\mu$ g/mL, 500  $\mu$ g/mL, or 1000  $\mu$ g/mL) resulted in a dose-dependent decrease in nitrite generation, increased production of superoxide, and decreased GSH levels compared to untreated cells, indicating ginger-induced apoptosis through the generation of ROS (Chen et al. 2007).

Ginger is also recognized for its potential usefulness to reduce nausea. To determine whether ginger had antiemetic effects in cisplatin-induced emesis, Manusirivithaya et al. (2004) conducted a randomized, double-blinded, crossover study in 48 gynecologic cancer patients. The addition of ginger (1 g/day) to a standard antiemetic regimen has no advantage in reducing nausea or vomiting in the acute phase of cisplatin-induced emesis. In the delayed phase, ginger and metoclopramide have no statistically significant difference in efficacy (Manusirivithaya et al. 2004). In another study, 1000 mg of ginger was compared to 20-mg intravenous (IV) metoclopramide, and to 4-mg IV ondansetron in controlling nausea in patients receiving cyclophosphamide chemotherapy. Ginger was determined to be as effective as metoclopramide, but neither was as effective as ondansetron (Sontakke, Thawani, and Naik 2003).

Overall, while the anticancer findings of ginger are intriguing and several processes may be associated with the observed responses, additional studies are needed to clarify the underlying mechanisms and to determine overall benefits to humans (Pan et al. 2008).



28 April 2016

The difference between gata vāta and āvarana ; swa sthānastha vāta helps to maintain the homeostasis or swāsthya but when avarodh to this gati takes place may be due to any reason the swa mārghāsthita vāta gets vimārga gata as explained in samprapti of śōtha (Ca. Ci. 12/8).

Various edemas are either due to excessive secretion (apāna vāyu) or reduced absorption (prāna vāyu) as understood in samprapti of udara. Disturbed concentration of solutes and solvents causes changes in pressure (vyāna vāyu) either intravascular or extra vascular. The electrolyte balance is brought about by sweda dōṣa ambu srotas sthāyi vāyu i.e. samāna vāyu. Five distinguished entities are Panchātmā vāta at one place .. Cellular description of Panchātmā vāta , pitta and kapha can help better understanding of ĀVARANA... In raktagata vāta, rakta dhātu gets vitiated by vāta dōṣa leading to shoshan of rakta dhātu; thus raktadhātu is unable to carry-out its normal function of jeevana, varnaprasādana, mānsa poshan etc. Vaivarnya is caused due to loss of varnaprasādana karma, due to depletion of mānsa poshana, krishata and tivra ruja (Ischaemic pain) is observed. CREST syndrome can also be understood on the basis of rakta gatavāta. In Sirāgata vāta, Ācharya have used two words mahati sirā and tanu sirā which resembles the two conditions related to vessels viz. aneurysm and narrowing of vessels. Aneurysms are a result of a weakened blood vessel wall. The repeated trauma of blood flowing through the vessel may contribute to degeneration of the vessel wall. Bleeding through the aneurysm may cause edema (śōpha). Pulsation (spandate) may be felt.

Narrowing of vessels mainly in the periphery is to be considered. Peripheral artery disease wherein there is no pulsation (suptā iti nispanḍā) and pain is observed. Intermittant Claudication, rest pain is the symptoms observed. Further due to reduced blood supply tissue loss (susyati) is also seen. Muscular atrophy manifests as sequel of Peripheral Arterial Disease. Raktāvṛta vāta are near to

some connective tissue disorders manifested with myalgia, insidious arthralgia, talengectacia and soft tissue inflammations.

Hetu explained in vidhishonitiya adhyaya are responsible for quantitative increase of rakta dhātu which impedes the gati of vāta dōṣa hence normal parivahana is hampered and stagnation takes place leading to sanga this is the reason why in rakta āvr̥ta vāta, rāga yukta śōtha, mandala, local dāha and vedāna have been explained. It can be compared with urticaria or vasculitis wherein there are rashes, burning sensation, pain, wheel and flare like presentation. These three above description show the difference between gata and āvr̥ta vāta..

5 Feb 2015

### Features of aavarana;

as per acharya panini agni vaayu mana are components of phonation.. praana vaayu as controller and samaana vaayu as stimulator of agni interplay (praana avrita samana) in moordhaa and manifests jad gadgada vaak n mookata...Since samaana is aavrita so there is no agni uttejana (acharya chakrapani ) and therefore vaayu ( praana) is not stimulated (prerita) and the coordination between mana vaayu and agni do not develop hence jada gadgad vaak and mookataa manifest..Kaayaagni means agni and sa prerayati maaruta.. the action of samaana is prerayati agni , ie aavrita means agni uttejana abhava and in turn kaayaangi becomes inefficient to activate vaayu to link with mana to induce phonation...Wernick's center - arcuate fasciculation - broca center - motor cortex - speech musculature - phonation.. wernick center is psychosensory center and broca center is motor center for speech..Since praana and samaana interplays in moordhaa , it may be wernick aphasia/ broca aphasia..Being psychosensory wernick's center is place for action of kaayaagni and mana and broca 's center and motor cortex are place for maaruta ( praana vaata ), efferent fibres , larynx and speech musculature are places for udaana karma.. here CNS lesions so praanaavrita samaana are referred.To act on samaana and praana \*chatusprayogah shasyante snehaah tatra sayaapana\* are indicated..Medhya dravya shrita ghrita like brahmi ghrita.. there is interplay between praana and samaana at higher center .. as described earlier with help of panini shiksha , praana as aavaraka and samaana as aavrita involve in loss or impaired speech.. shoshan by samaana may be one reason to induce dysphonia/aphonia at periphery ( lungs and larynx) level.. Dysphonia manifests in severe dehydration.. but in this reference , CNS pathology is cause of aphasia.. acharya kashyap mentioned tatra vaagindriya tvekam dvividhaa bhinnam yathaa karau... (phakka chi 7-9) .. it also indicates towards Sound - organ of corti - hearing center - wernick 's center ( psychosensory) - arcuate fasciculi - broca's center - motor cortex -efferent

nerve fibres - speech musculatures - speech.. Due to praanavrita samaana , Wernick aphasia with decreased mental acuity( MR) can be reffered..

How does one become aavaraka and one aavrita ? means pathological relationships develop in which the hetu vitiate the dosha as aavaraka and same hetu decrease the action/ strength of an other dosha , aavrita dosha.. eg.. rooksha gunaत्मका aahaar will impair the action of samaana , so agni karma is reduced and rookshata also leads to hard and less stool formation and in turn evacuatory actoin of apaana becomes less , so kitta part of aahara retains for long time . Therefore grhani gulma aamaashaya shoola hridroga like diseases are consequences of samaana aavrita apaana..As i think there must be interplay between dominant and dominated dosha on basis of interlinked etiopathogenesis.. see in vyaana aavrita apaana and apaana aavrita vyaana , urdhva and adho gati of apaana in relation to vridha vyaana and hraasita vyaana manifests vamaana and atimalapravriti respectively.. Urdhva gati of apaana ( aavrita) in relation with udaana ( aavaraka) manifests chhardi and shvaasaadi roga , as it becomes udaana bhava prapanna..

tathaa apeedam amoortatvamakathinavaachakam na tu avayava pritishedhakam ; ten vayoh vayu antarena gatihanana roopam aavaranam upapannamevah.. drishta cha vaayunaa vayu antarena upahatena vaatakundalita bahirapi , ten upapannama aavaranam vaataanaam parasparam.. All these show vaayusya frictionless action/apratighaatatva/avyaahata/aparityakta gati. So maitaining normalcy of organ-system.. Homeostasis.. ayusho anuvriti pratyaya bhuto bhavati akupitah.. ch.su.12..its akathina (not kathina) means absence of hardness/compactness, simply frictionless... i think sookshma , sookshmatara and sookshmatama nature of vaayu are reasons of anyonya aavarana.. eg..sooksma may be aavaraka for sookshmatara and sookshmatama , sookshmatara may be for sookshmatama vaayu.... its relative sookshmatara in between vaata bheda leading to impairment of gati of both aavaraka and aavrita dosha..



1 Feb 2015

**Vyaana aavrita apaana** ; There is vaman aadhaman udaavarta gulma and parikartikaa in vyaanaavrita apaana.. the urdhvagati of apaana induced by severity of vyaana leads to such diseases in consequence.. eg.. more severely vitiated vyaana can do vamaana due to max urdhvagati of apaana and less severe vyaana can affect on apaana only in anus so parikartika manifests. It is interplay between both with predominance of vyaana , but cant ignore gati vaishmya of apaana.. Gulm in this reference means vyaana is obstructing the passage of apaana and apaana becomes pratiloma and in turn gulm occurs.. 5+ vyaana - vamaana , 4+ vyaana - adhamana , 3+ vyaana -udavarta, 2+ vyaana - gulma and 1+vyaana-parikartika, The extent of vitiation of apaana is constant ..or vyaana is constant and urdhvagati of apaana becomes 5+,4+, 3+, 2+, and1+ to induce same features vamaadi in sequences , futher study is required.... In anyonya aavarana multiple diseases are observed due to gati vaishmya of both aavaraka and aavrita..I think vamaana in vyaanaavrita apaana may be due to colonic obstruction , if fecal odour of vomitus is present.. In one case of hemianopia there was no obvious reason observed , that i thought praanaavrita vyaana..Site / location of happening of aavarana also can cause variable features..

23 May 2013

Vyane pittavrite tu syaddahah sarvang klaham. Gatra vikshep sangah( gatravikshepanoparamah) cha sasantapah savedanah.ch.chi.28/227-228... polyneuropathy..

20 Feb 2016

**Acharya charak, Acharya Chakrapani , and modern review : Annāvrta Vāta:**

Āhar when taken in excess the prokinetic movement is reduced and the āhar is not propelled forward leading to stretch reflex. The pain of obstruction of hollow abdominal viscera is classically described as intermittent food related abdominal pain followed by remission.

Mūtra-āvrta vāta: These symptoms are seen in mūtravega dharan. Normal urine formation takes place but the patient does not evacuate it timely leads to the avarodha of vāta gati. Vāta is unable to contract the detrusor muscle thus there is mūtra apravriti and inturn bladder distension. This condition may also arise in neurogenic bladder.

Atonic bladder – Micturition reflex contraction cannot occur if the sensory nerve fibres from the bladder to the spinal cord are destroyed, thereby preventing transmission of stretch signals from the bladder. When this happens, a person loses bladder control, despite intact efferent fibers from the cord to the bladder and despite intact neurogenic connections within the brain. Instead of emptying periodically the bladder fills to capacity and overflows a few drops at a time through the urethra. This is called overflow incontinence. Crush injury is the common cause.

Purishāvrta Vāta: Dietary fibres adsorb water and this increases the bulk of stools and helps reducing the tendency to constipation by encouraging bowel propulsive movements. Diet low in fibres content reduces the healthy bowel movements. Stools are formed but due to slow transit there is hard and peltly stool formation which finds it difficult to pass out.

Malavega dharan may also cause the above symptoms. In Diabetes mellitus whenever there is neurogenic involvement, peristalsis are reduced creating the above symptom. Spastic colon may also be considered.

23 May 2013

Bhukte kukshau cha rook jeerne shamyati annavrite anile..ch.chi.28/69- Gastric ulcer..

2 Feb 2015

Anna aavrita vaata ; bhukte kukshau cha rook jeerne shaamyati..(ch.chi 28/ 69).. Abdominal pain occurs after intake of food and relieved after digestion.. after heavy , spicy meals abdominal pain occurs, if intragastric volume is increased either due to quantity and volume of food or due to excess gastric juice secretion or both. Its dyspepsia, since as treatment \* annaavrite tat ullekha (vamanam iti paatha) paachanam deepanam laghu \* are indicated.. by vaman , aahaara is vomited out to decrease intragastric volume hence pain subsides.. in gastric ulcer (for differential diagnosis ) pain occurs after food and relieved after emptyness of gastric contents into duodenum , but in presence of ulcer if induced vaman is done as therapeutic regimen it can lead to perforation.. vaman in gastric ulcer is harmful . Pain after food develops aversion to food ie annadvesha and in turn patient start to loose weight, especialy in GU... in such condition daadimaadi ghrita shataavari ghrita are good choice... Sitophobia ( aversion to food ) is one feature of abdominal tuberculosis , here other features help in differential diagnosis...

2 June 2013

Majjaavrite vinaamah syat jrimbhanam pariveshtanam. Shoolam tu pidyamaane cha paanibhyam labhate sukham.. ch.chi.28/67-68. Majjaavrit vat-> spinal canal stenosis..

**शुक्रावृत वात in śukrāvṛta vāta** immature sperms are formed which ; lose their forward movement activity. Ciliary dyskinesia (Kartagener Syndrome) can be included in this group. Since motility is reduced

it leads to infertility. Y chromosomes microdeletions and POLG variants are increasingly recognised as a cause of azoospermia or oligospermia. Primary gonadal deficiency with low-testosterone and decreased spermatogenesis are the reason for infertility

Patients with normal hormonal levels and low sperm count may be found in obstructive anomaly of vas deferens and epididymus. shukrala and shukravirechaniya dravya are indicated..

### **Rakta avrita Vata**

Vata dosha alongwith ruksha, laghu, shita, khara, suksma guna possesses cala guna.

Yasya prerane shakti sa cala .Va.Su.1/40 (Arundatta)

Cala guna is essential for avyhatgati of vata dosha and also for the pravartan (stimulation), chesta (movement), vyuhakar (organizing), ksepta (excretion), santan gati (sinus rhythm), pumping and pulsation. Thus whenever cala guna will be affected vikshepa, samvahana, parivahana, chavan, pargaman, sravan, visyandana karma will be hampered.

As understood in grahani adhyaya yugpat, sarvatra, continuous vikshepa of rasa rupa dhatu is by vyanvayu which is responsible for nutrition of sthyayi dhatu. Whenever due to kha vaigunya the vikshepita rasa gets obstructed/ stagnated, pathogenesis takes place. Thus for avyhatgati of vata normalcy of marga and margastha dhatu is essential.



Marga means various channels, srotas, sira, dhamani, rasayani, rasavahini, nadi, pantha, sharirchidra, aashaya, niketa etc. whereas margastha dhatu means drava rupa asthayi raktadi dhatu.

Specific ratio of pancamahabhut maintains the dravatva of dhatu. More the viscosity slow is the flow of the drava rupa dhatu. Viscosity will increase whenever parthivata will increase in proportion in margastha dhatu. Change in specific proportion is primarily due to agni. It may be at level of jatharagni, dhatvagni or bhutagni. Secondly, anupahat agni is responsible for maintaining of normalcy of dhatus. Agnimandya leads to apachit dhatu vridhi. Such apachit dhatu are nothing but aam which may act as antigen. Vyadhi vighatkar bhava comes in action to prevent adherence of aam with specific dhatu.

Thus presence of aam and vyadhi vighatkar bhava changes the specific ratio of dravatva leading to reduction of flow (Saratva) or capillary perfusion and increase in organ congestion and syndromes of hyperviscosity.

Therefore upahata agni causes variation in dravata leading to obstruction or aavarana of vata dosha causing aavrita vata.

Secondly anupahat srotas is necessary for dhatu poshan. Srotas over here means marga or channels. If the patency of channels is hampered it disturbs the flow of drava rupa dhatu. Patency of channels depends on bija (genetic), environmental factors (external factors) and internal environment within the channels.

It is important to understand the significance why Kush Sankrityayana while explaining 6 qualities did not explain kala as quality of vata whereas he explained daruna as quality of Vata. Secondly answer given to question of Kankayan Rishi on how asanghata or amurta vata gets prakopita or prashaman by murta dravya.

Acharya Badish Dharmargava has explained that ruksha, laghu, shita, khara, vishada, sushira and daruna guna acts on sharir and as vayu takes ashraya of sharir for its activity prakopa of vayu takes place whereas snigdha, guru, ushna,

slakshna, mridu, picchila guna when increase in sharir they do prashaman of ashrayi vata dosha. Therefore darunata i.e. hardness of marga causes prakopa of vata. Specific hardness of channels is essential for normal flow. If such darunata is lost it causes aneurysm leading to impaired flow of drava rupi dhatu as understood in siragata vata whereas hardness when increased the flow is obstructed causing vataprakopa.

Thus one can conclude from above discussion that for normalcy of vatagati following entities are essential.

1) Specific ratio of drava rupa dhatu ( Viscosity)

2) Marga ( Channels)

Aahar

|

Agnimandya

|

Apakva aahar rasa

|

Dhatusma (Upahata ushma)

|

Sama dhatu [asthayi / dravarupa]

|

Vikara vighata bhava if vighatkara bhava Vyadhiutpatti without aavarana

are unable to regularize

|

Specific ratio of dravata is altered / bija dusti and/or environmental factors

|

Viscosity rheological forces

Loss of patency of channels

|

Yatra sanga kha vaigunya

(upahat srotas)

|

Vyadhiutpatti without aavarana

|

Avarodha to avyaha gati

Inflammation of channels

|

Aavrita vata [upahat srotasgata vayu]

|

Vyadhi utpatti with aavarana

|

Rakta Avrita Vata

Raktavrite sa daha arti tvak mansa antarya bhrisam |

Bhavet sa raga swayathu jayante mandalani cha ||

Ca.Ci.28 / 215

In rakta avrita vata there is either quantitative and /or qualitative increase in rakta dhatu which obstructs the gati of vata dosha leading to aavrita vata.

In Vidhishonitiya adhyaya Caraka has explained rakta dustikara hetu which are cause for qualitative and quantitative impairment of rakta dhatu.

Ati lavan rasa sevan causes quantitative increase of rakta ( Raktam Vardhayati)

Kshar causes pachana, daran of the srotas.

Amla rasa does pachana, mansa vidaha ( lepana karma is of mansa and vessels are made up of muscle fibers) and swayathu utpadayati. Rakta dusti, causes inflammation.

Katu rasa reduces bala and has quality to irritate the mucosal lining.

Kulatha has ushna virya and amla vipak. It causes amlapitta and thereby after vitiating pitta causes rakta dusti. Kulatha is mentioned hetu in raktapitta where there is quantitative increase of rakta.

Masha although balya, when taken in excess quantity causes mala vridhi and is ushna in nature.

Tila taila, mulaka, pindalu, jalaja and anup mansa by their ushna guna causes raktadusti.

Sura, souvirak, sukta are ushna and are raktadustikara.



Virudha, upaklinna anna, puti anna, diva swap are agnimandyakar and have low nutritional values.

Aatap and anala sanyog are external factors which directly affect the small blood vessels and are cause for local pathogenesis. Similarly abhighata/ injury also causes raktadusti as seen in case of superficial venous thrombosis after catheterization.

One thing is common that all the above ahariya dravyas are vatashamaka therefore when taken in excess will hamper gati of vatadosha.

The quantitative increase of rakta causes increase in viscosity and thereby hampering the gati of vata as seen in cases of polycythaemia rubra vera.

POLYCYTHAEMIA RUBRA VERA (PRV) is a myeloproliferative disorder wherein there is an abnormally high number of red blood cells with or without abnormally high number of platelets and WBC, because of the circulation of extra number of blood cells, blood becomes thicker or more sludgy than normal causing blood to flow slowly and giving rise to certain symptoms and also increasing risk of thrombosis.

Over 19 in 20 people with PRV have an abnormality (called a mutation) in a protein called the JAK2 protein. The JAK2 protein normally helps to regulate and control the productions of blood cells.

The sludgy blood flow means oxygen cannot get to the tissues leading to various symptoms like headache, chest pain, pain in calf muscles (arti tvak mansa antarjo bhrisam), tiredness, dizziness, tinnitus, blurring of vision.

Further 4 out of 10 patients release histamine causing itching and urticarial rash (sa raga swayathu jayante mandalanicha). Complexion is a bit more ruddy than normal due to increase number of RBC.

Some patients may present with bruising, epistaxis, gastrointestinal blood. This presentation should be understood under raktapitta. Also about 1 in 10 people with PRV develop gout wherein concept of vatarakta should be considered.

This also signifies the common hetus in raktapitta, vatarakta and raktadusti.

Venesection is one among the treatment for PRV which removes the extra red blood cells and make blood less viscous so that it circulates better. Regular venesection is preferred. Acharyas have explained raktamokshana in vatarakta and treatment of raktavritta vata is similar to vatarakta.

Apparent erythrocytosis where RBC are more concentrated can be caused by many things such as obesity (recollect the jalaj, anup, mansa, masha etc are cause for sthaulya and raktadusti), alcohol (sura sauvira), stress (krodhadi), smoking (tikshna ushna dravya), less fluid, diuretics, high blood pressure, kidney disease etc. The presentation is similar to PRV.

Hyperviscosity Syndrome; it is a group of symptoms triggered by increase in the viscosity of the blood

Type of hyper viscosity syndrome vary by pathology,

- 1) Serum hyperviscosity which may cause neurologic or ocular disease.
- 2) Polycythemic hyperviscosity which results in reduced blood flow or capillary perfusion and increased organ congestion.
- 3) Syndrome of hyperviscosity caused by reduced deformability of RBC often evident in sickle cell anaemia (note pinyaka, harita shaka etc have low nutritional value (vit B12) are explained as rakta dustikar hetu)

Blood viscosity is a measure of the resistance to the flow of blood. This biophysical property makes it a critical determinant of friction against the vessel walls, the rate of venous return, the work required for the heart to pump blood and how much oxygen is transported to tissues and organs. These functions of the

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cardiovascular system are directly related to vascular resistance, preload, afterload and perfusion respectively.

The primary determinants of blood viscosity are haematocrit, RBC deformability, RBC aggregation and plasma viscosity. Plasma viscosity is determined by water content and macromolecular components, so the factors that affect blood viscosity are the plasma protein concentration and types of proteins in the plasma. Elevation of plasma viscosity correlates to the progression of coronary and peripheral vascular diseases. Many conventional cardiovascular risk factors and outcomes have been independently correlated with whole blood viscosity. Hypertension, total cholesterol, LDL, VLDL, triglycerides, diabetes, metabolic syndrome, obesity, male gender, aging, cigarette smoking have all been positively linked to whole blood viscosity.

PHLEBITIS is the inflammation of vein. It most commonly occurs in superficial veins. Phlebitis often occurs in conjunction with thrombosis and is then called as thrombophlebitis.

Phlebitis is typically caused by local trauma (abhighata to the vein). Phlebitis can also result from certain medications and drugs that irritate the veins i.e. vascular irritant (tikṣṇa, uṣṇa, kṣhara katu dravya). These causes are directly affecting the marga or the channels / siras. Immune factors (Vikar vighatkar bhava) come into play at the site of injury, if they are strong enough no inflammation occurs but if there is faulty response inflammation occurs at the site of injury (kha vaigunya). Inflamed endothelium now leads to coagulopathy. Thrombus signs does formed causes obstruction to the flow of blood leading to symptoms like localized redness and swelling (sa raga swayathu mandala), pain or burning along the length of the vein ( sa daha arti tvak mansa antarja bhrusam) and veins become hard and cord like ( darunta).

ERYTHROMELALGIA is a rare neurovascular peripheral pain disorder (arti) in which blood vessels, usually in the lower extremity or hands are episodically blocked

frequently (on and off daily) then become hyperemic and inflame (rasa, swayathu). The attacks are produced and are commonly triggered by heat, pressure, mild activity, exertion, insomnia or stress.

It is classified into two primary and secondary. The primary type resembles the second phase of vatarakta wherein vitiated vata dosha impedes gati of rakta whereas in secondary erythromelagia very specially caused due to essential thrombocytosis resemble rakta avrita vata.

Conclusion: Rakta avritta vata is a process of pathogenesis wherein raktavridhhi (quantitative increase of rakta) impedes the gati of vata which leads to symptoms like pain, redness, burning sensation and localized inflammation. The symptoms can be observed in various diseases like erythromelalgia, phlebitis, PRV and apparent erythrocytosis. Thus raktavritta vata is not a single disease but initial factor of pathology.

Raktamokshana reduces quantitative increase of blood. Virechan is the best shodhan procedure for raktadusti. Fluid loss due to virechan also has impact on intravascular quantity of fluid plasma does reducing the avarodh.

Shita pradaha mainly medicines like dashanga lepa, kamala, ushira, yastimadhu, sariva, chandan, padmak, darbha etc drugs helps to reduce local inflammatory response. Anti thrombotic effect of darbha, kamala also reduces the avarodh to the gati of vata.

Medicines which reduces avarodh of rakta and quantitative increase of rakta alongwith which reduces inflammation of siras is helpful in rakta avrita vata.



23 Feb 2016

### **ANUKTA ĀVARANA –**

My approach ( 8 types of anyonya avarana are not described , just, their name is referred in charak samhita ) , here , explained with modern perspective.. !

#### **Vyāna Āvrta Udāna**

Vyāna is associated with gati and prakshepan while udāna is associated with bala prayatna and urjā. Vikrut vyāna has impaired gati which when impedes udāna will reduce the bala, prayatna ādi karma of udāna.

Sympathetic fibres originate in the hypothalamus, pass down the brain stem and cervical spinal cord to emerge at T1 level, return back up to the eye in association with the internal carotid artery and supply the dilator pupillae. Lesion in the sympathetic pathway cause Horner's syndrome. The reason may be central (at the level of Hypothalamus / brain stem) or at the periphery (at the level of lung apex, carotid artery) or may be idiopathic.

Vyāna āvrta udāna can also be considered in paroxysmal tachycardia. Abnormalities in different portions of the heart including the atria, the Purkinje system, or the ventricles, can occasionally cause rapid rhythmical discharge of impulses that spread in directions throughout the heart. This is believed to be caused most frequently by re-entrant circus movement feedback pathways that set up local repeated self re-excitation. The above process occurs unless considerable ischemic damage and may lead to ventricular fibrillation. Thus there is never a coordinate contraction of all the ventricular muscle at once which is required for cardiac pumping. Patient may complaint of palpitation or symptoms such as dizziness, dyspnoea, fatigueability i.e. bala, prayatna are reduced.

#### **Apāna Āvrta Samāna ;**

Apāna is responsible for srijan karma. Vikrita Apāna increases the nishkraman prakriya. Increase Hustration reflex causes excessive propulsion movement. Excess motility causes reduced absorption. The body is unable to reabsorb bicarbonate ions i.e. Samāna karma is reduced. Loss of bicarbonate causes rise of H<sup>+</sup>. Body compensates the process by increased ventilation. The PaCO<sub>2</sub> is reduced secondarily by hyperventilation which mitigates the rise in H<sup>+</sup> leading to metabolic acidosis.

Diarrhea associated with passage of more than 200g of stool with urgency of defaecation and faecal incontinence. This may lead to malabsorption leading to hypoalbuminaemia, hypocalcaemia and vitamin D deficiency, hypomagnesaemia, phosphate, zinc and weight loss.

#### Prāna Āvṛta Apāna

Prāna vāyu function is associated with controlling system of the body, as said by Nyāyachandrikākār. Prāna vāyu helps in assimilation and maintain homeostasis.

Apāna is responsible for elimination. Considering pakvāshaya it may be compared with srijan of purisa mūtra etc. and at cellular level function of apāna is removal of cellular products within the cell. In this particular condition of prāna āvṛta apāna the vikrita prāna obstructs the gati of apāna and it is unable to release the cellular products. This can be understood in condition of Brainstem lesion where in the control over CO<sub>2</sub> expiration is lost. Depletion of CO<sub>2</sub> expiration leads to increase in concentration of CO<sub>2</sub> in blood resulting in respiratory failure of Type II origin i.e. severe respiratory acidosis. A simple sleep apnoea / hypopnoea syndrome may also be considered.

अनुक्त आवरण4,.

#### Vyāna Āvṛta Samāna

There is interplay between gati of vyāna with gati of Samāna. Therefore when rasa vikshepan karma of vyāna related to Samāna vāyu is vitiated, the later becomes āvrta and in turn annapācana, vivechan and munchan karma of Samāna are inhibited or decreased.

Sympathetic nerves have dual action in some cases; it increases secretion but if parasympathetic is already causing copious secretion than sympathetic usually reduces the secretion mainly by vasoconstriction reducing the blood supply.

Although Enteric Nervous System (ENS) can function autonomously; Autonomic Nervous System (ANS) connects ENS centrally. When ANS activity is increased it has its impact on gastrointestinal tract. Sympathetic overstimulation causes vasoconstriction which reduces secretion of gastric juices and pancreas exocrine secretion. Their insufficiency can cause malabsorption syndrome in which predominant feature is steatorrhoea, deficiency of fat soluble vitamins, protein and carbohydrate deficiency related features.

As compensatory mechanism vasodilatation in skin leads to excessive sweating and skin related features.

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1 July 2014

In pittavrit samaana acharya charak mentions upaghaatah tatha ushmanah. (acharya chakrapani) iti atra pittena api aavrite samaane agni uttejanaa abhaavaat ushmana upaghaato geyah.. ch.chi 28/226..the same can be observed in Zollinger Ellison's syndrome ; excess HCl secretion - acidic intestinal Ph - no activation of pancreatic zymogens into active enzymes-indigestion...t/t pittaghna drugs ; tikta pradhaana which also activates samaana vaata

अनुक्त आवरण Udāna Āvr̥ta Samāna; (5) -

In this particular condition as udāna vikriti takes place anabolism increase reducing the catabolism. This is observed in Hypothyroidism where in weight gain is seen with decreased appetite.

Leptin is secreted by adipose cells and acts primarily through the Hypothalamus. Its level of production provides an index of adipose energy stores. High leptin levels decrease food intake and increase energy expenditure. The OB gene is present in humans and expressed in fats. The obesity in these individuals begins shortly after birth, is severe and is accompanied by neuroendocrine



abnormalities. Role of central hypothyroidism has been understood in mouse model.

Another condition may be considered where increased acetylcholine stimulates increased ATP (urjā) which further increases excessive secretion of fluids and electrolytes in addition to normal viscid alkaline mucus which further increases gastrointestinal activity causing reduced absorption (annasoshan, vivechan karma) leading to malabsorption diarrhea.

Anukta avarana ( 6,7,8) ; Samāna Āvrta Prāna

Role of Samāna along with agnideepan is to help in pācana and sāra kitta vibhajan. Thus along with pitta, Samāna plays its major role in metabolism. If Samāna gets vitiated then sāra kitta vibhajan does not takes place properly and kitta bhaga gets upashoshit along with sāra bhaga. Thus kitta munchan prakriyā does not take place. This condition may be noted in metabolic acidosis, hypercalcemia and uraemia. The kitta bhaga now alters the gati of prāna or in other words neuronal excitability. It may show symptoms like confusion seizures, coma and death. It may also depress the respiratory centre causing hyperventilation or Kussmaul breathing.

Samāna Āvrta Udāna

The same kitta bhāg depending on sthān obstructs the gati of udāna. The bala, prayatna, urjā ādi karma are reduced.

Conditions can be observed in hepatic coma where in increase levels of ammonia (kitta bhāg) interfere with cerebral energy metabolism and with Na<sup>+</sup>, K<sup>+</sup>, ATPase pump. Number and size of astrocytes are increased. They alter the nerve cell function and causes symptoms of fatiguebility, altered sensorium and coma. (prayatna nāśa).

Similarly Hyperthyroidism may also be considered. Samāna is said to be agnibala pradha, it leads to increase in catabolism. Energy gets exhausted with increase catabolism reducing the bala prayatna which is the role of udāna vāyu as seen in thyrotoxicosis.

#### Apāna Āvrta Prāna

Interplay exists between apāna the eliminator and prāna the controller of the body system. Mismatching between apāna and prāna karma leads to various disorders. If the srijan karma and gati of apāna related to ādān karma of prāna gets vitiated the prāna vāyu gets āvrta and in turn causing difficulty in breathing, confusion, coma and death.

Loss of Na<sup>+</sup>, Cl<sup>-</sup>, H<sup>+</sup> and extra cellular fluid depletion occurs in excessive administration of diuretics or in congenital chlorodiarrhoea. It leads to increase concentration of plasma HCO<sub>3</sub> which leads to condition such as apathy, confusion and drowsiness.

In anxiety induced hyperventilation excessive loss of CO<sub>2</sub> takes place. PaCO<sub>2</sub> and H<sup>+</sup> falls. The low PaCO<sub>2</sub> results in reduced renal Na<sup>+</sup>/H<sup>+</sup> exchange due to which patient feels short of oxygen.

I posted my approach to ANUKTA ĀVARANA before you , this hypothesis needs validation and mass acceptance .. I will like if some discussion with scientific approach is presented.. Acharya charak explained 12 types of anyonya avarana in details and left remaining 8 for future prospectus.. So , it's our duty to go through the literature review and make hypothesis helpful for therapeutic excellence.. thanks..

1 April 2016

### **Pakvaashayagata vaata ;**

The clinical features are aantrakoojana , shoola , aadhmaana , aanaaha , mootrakrichchha malakrichcha and trikavedanaa . if rooksha , laghu , Khara guna pradhaan aahaara will be consumed continuously , katu paakaavashthaa will last longer and katu bhaava will be produced excessively.. All these happen in pakvaashaya , so prakupita vaata resides in svasthaana ie pakvaashaya and leading to increased transit time , hence time for absorption is increased , already in aahara snigdhaansha is very less , and that also absolutely absorbed due to increased transit time , therefore shushka , alpa mala is formed , and during passage of mala shoola and difficulty manifest , rooksha aahaara have less water composition , therefore decreased urination with difficulty occurs .. Prakupita vaata in pakvaashaya manifests aantrakoojana aadhmaana and aanaaha like features ; reason is vishtabdhathaa.. Similar features appear in gulma , udaavarta and purishaavrita vaata ; shows that vaata gati is hampered.. As we know that for normal vaata gati a due amount of snigdhaansha is needful. Trika vedanaa is presented due to difficulty in defecation.. Snigdha anulomaka dravya are indicated ; further shows that rooksha is predominant hetu ..Vishtambha ( retention ) of vaayu (flatus/gas) , mala ( fecal matter ), and mootra ( urine ) manifests above mentioned symptoms.. Similar happens due to apakva aahaara but in form of alasaka.. ( aama pradoshaja vikaara )..Very thin line difference is between pakvaashaya gata vaata , guda gata vaata , udaavarta and purishaavrita vaata ; in all these , treatment is as per udaavarta.. Then , question comes , why all these different entities are classified , if treatment is same ? For perfect understanding of disease process , because few different symptoms are observed in all above mentioned conditions.. Approach to patient is important for proper diagnosis and treatment.. What's about hetu sammuchaya ? Since vaata is in pakvaashaya , so , I expressed more importance of aahara.. If vaata goes to rakta maansa and ashthi , I will explain both aahaara and vihaara. In Aamaashaya gata

vaata , kosktha gata vaata , maanasika hetu may be seen predominantly. Congenital disease like megacolon may lead to sanga or aavarana.. Nothing to vaata primarily , but secondary.. Here , in pakvaashaya gata vaata , vaata prakopaka is primary and initial factor.. Treatment plan ; Diet modification, adequate water intake , snigdha anulomaka dravya..

26 June 2014

In case of guda gata vaata there is ashma sharkarah ( ch.chi28/26.. charaka mentions " guda pakvaashayastho tu karma udavartanudhitam.(ch.chi28/90)... i had a patient of enterolithiasis due to over use of ground nut.. i used gandharva haritaki agnitundi vati and sinhanaad guggul.. all stones were eradicated .. Acharya charak mentions ; prashastama erandajam tailam ( ch.chi26/29).. yaduktam ; sthaanam jayeddhvi poorvam...

18 Feb 2016

Mānsa meda dhātu have similar characteristic both being snigdha, guru, sthira guna pradhan which gets vitiated by rūkṣa, laghu and cala guna of vāta leading to disorder called mānsamedogata vāta. Various myopathies can be included under mānsamedogata vāta specially Carnitine palmitoyltransferase deficiency in which severe pain with fatigueness is seen. Myasthenia gravis can also be considered in mānsamedogata vāta.

Asthigata vāta is multiple clinical conditions in which osteoporosity are a marked feature and majjāgata vāta is the same associated with marked synovitis. Due to external injury or due to pressure the asthi and majjā dhātu gets deranged leading to pain mainly at asthi parva or at the level of joints. The pain is continuous and it may later on show periarticular muscular atrophy as its late complication. It can be collectively understood under osteoarthritis where in focal loss of articular hyaline cartilage is seen with simultaneous proliferation of new bone with remodelling of joint contour (sclerosis).



23 may 2013

Guru angam tudyate ati artham dandmushti hatam tatha. Sarook shramitam ati artham (undue fatigability) mansmedogate anile.. ch.chi28/32..Myopathies , or skeletal muscle diseases

26 June 2014

The difference between rakta aavrit vaata rakta gata vaata and vaatarakta...In raktaavrit vaata rakta is aavaraka and vaata is aavrit.. so initial cause is to vitiate rakta enough to obstruct gati of vaata... as it can be seen in deep venous thrombosis ..

In rakta gata vaata , initially vaata prakopa occurs and leading to vitiation of rakta... in vaatarakta vaata and rakta dushti separately followed by margasyaavarana by dusht rakta.. 3 stages of samprapti of vaatarakta..Acharya charak mentions different clinical features in raktagata vaata and raktaavrit vaata so its different entities..

In raktapitta there is rakta dushti but initial factor is pitta prakop leading to raktadushti , raktavridhi - raktapitta... e.g. amla rasa - pitta prakopa -rakta dushti , lavana rasa - pitta prakopa - rakta vridhi , katu rasa - pitta prakopa -rakta sanghat bheda ( thrombolytic/ anticoagulant )....ch.su. 26.. it means when all these rasa are used in excess quantity for long term, raktapitta occurs.....Hetu -samprapti - lakshana -chikitsa are interrelated , so they must be well explained with references to make understanding easy....Abhighaata (trauma) - vaata prakopa - raktagata -tivra rooja sasantaapaa vaivarnyam aroonshi etc.. (inflammation). Ruksha aahaara -vaata prakopa-rakta gata -decreased tarpana - krishataa etc....Long standing - rakta dushti - grathita rakta utapatti -avarodha- aavrit vaata- shvathu and mandala with daaha & raaga - DVT

9 April 2016

### **My approach to parkinson's disease ;**

Since Parkinson's disease is neurodegenerative disease , so we have to think dhaatukshaya janya vaata prakopa.. The failure of dopamine receptors or deficiency of dopamine neurotransmitter is main reason for clinical features , but due to dopamine deficiency , the exaggerated acetylcholine also play roles in pathophysiology of features like tremors , therefore , anticholinergic & antihistamine like trihexiphenyldil is found effective , however levodopa , dopamine agonist , with carbidopa is first choice.. The issue is first pass metabolism of dopamine in natural resources . more than 90 % of dopamine is destroyed in periphery and reach very minimum in brain. Carbidopa inhibits peripheral metabolism of levodopa , so , most of levodopa crosses blood brain barrier and reach to basal ganglia and surrounding areas to provide dopamine..

Ayurveda perspective ; tremors ( increased chala guna ) , bradykinesia & hypokinesia ( decreased chala guna ) , , rigidity & postural change , short and shuffling gait ( increased rooksha , sheets guna ) , etc are features due to vaata prakopa.. Praana , udaana and vyaana vaayu bheda are effected predominantly.. Snehana , mridu svedana, shirodhara , anuvasaana , yaapanaa basti look to work on rooksha , chala , sheeta guna of vaata, and they work , but result does not last longer. Vaataghna drugs like vrihatvaat chintamani rasa , yogendra rasa , etc also have limited roles in PD . in few cases , I found result with sinhanaada guggula , mahayogaraja guggula , and the combination of ashwagandha bala shatavari kapikachchhu chopachini maasha and dashamoola with ghrita , abhyanga by mahaamaasha tail , mridu naadi sveda ( not more than 5 mins ) shirodhara by kvaatha of ashwagandha bala shatavari kapikachchhu chopachini maasha brahmi shankhapushpi aamalaki and dashamoola mixed with mahanarayan tail . yes , result is very good , if I continue levodopa etc prescribed by neuro physician with my treatment .. We can minimize on & off phenomena .. By reducing rigidity , betterment of life. We can improve gait , posture , speech , balance and cognitive

processes.. Result on tremors is there , but not so marked as in other features.. All these are my personal experience.Ghrita helps lipid soluble substances to cross blood brain barrier.. Ghrita kalpa of kapikachchhu etc may help in controlling PD.. Because of dopamine deficiency , vaata becomes vyaahata , parityakta from the svasthaana , therefore , the choice is to achieve avyaahata / aparityakta gati of vaata , ie possible only after supplementing dopamine into brain ( prakritisthaapana ).Future prospectus ; to work on PD with ayurveda perspective to provide better treatment than management of contemporary science..

{{One drug with multiple therapeutic response ; Vidanga is referred as triptighna , krimighna, kushthaghna , and shirovirechanopaga...

Yashtimadhu is indicated in jeevaneeya , shonitasthaapana , angamarda & daaha prashamana , mootra virechaniya , aashthaapanopaga , vamanopaga , snehopaga , kandughna , kanthya , varnya , sandhaaniya.. it's used in 15 formulations mentioned in Vaatarakta chikitsa..Such enlistings will help to understand how to select the combination for specific clinical entity/ disease..+}}

7 Feb 2016

**Dementia in ayurveda perspective** ; Dementia, a syndrome with many causes , is defined as an acquired deterioration in cognitive abilities that impairs the successful performance of activities of daily living. Memory is the most common cognitive ability lost with dementia. Language , visiospatial ability , calculation , judgement , and problem solving mental faculties are also affected.. Neuropsychiatric and social deficits also arise in many dementia syndrome, resulting in depression, apathy , hallucinations, delusions, agitation, insomnia, and disinhibition. Most patients with Alzheimer's disease begin with memory impairment , although in other dementias , such as frontotemporal dementia ,



memory loss is not present. Dementia results from the disruption of specific large scale neuronal networks ; the location and severity of synaptic and neuronal loss combine to produce the clinical features. Behavior and mood are modulated by noradrenergic , serotonergic , and dopaminergic pathways , whereas cholinergic signaling is critical for attention and memory functions. The dementias differ in the relative neurotransmitter deficit profiles ; accordingly , accurate diagnosis guides effective pharmacotherapy..The single strongest risk factor for dementia is increasing age.. The combined study of unmaada and vaata vyaadhi help to draft ayurveda principle in dementia..the characteristic microscopic findings are neuritic plaques and neurofibrillary tangles.. These changes lead to avarana , especially concerned with praana and vyaana ; leading to decrease in cortical levels of several proteins and neurotransmitters. Vaata , especially praana is regulator of mental , motor , sensory , and social functions.. Vyaana is responsible for rasaraktadi vikshepana , udana is for prayatna , karma , balaadi , samaana for local metabolism and apaana for clearance of accumulated undue substances.. Praana aavrita vyaana , udaana aavrita praana , samaana aavrita vyaana and praana aavrita apaana can be seen in different dementia.. Since it's syndrome , so multiple etiopathogenesis is present.. Treatment plan is variable depending on specific avarana. Praana is crucial to manage with help of keeping normal udaana, samaana , vyaana and apaana.. Harshana , aashvaasana , urdhvajatru karma , yapana basti , deepaniya ghrita , different rehabilitation program , vaataghna , balya , medhya , anulomaka , Rasayan like Brahma Rasayan , shilajita , guggula with milk are choices..

9 Dec 2014

Peripheral artery disease or dhamanigata vaata; Atherosclerosis or dhamanipratichay is major cause.. cold intolerance , numbness tingling , muscular fatigueability, muscular cramps/pain. On physical examination; decreased or absent pulses distal to obstruction , presence of bruits over narrowed artery ,

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muscular atrophy.with more severe disease , hair loss thickened nails smooth n shiny skin reduced skin temperature and pallor or cyanosis of affected part ( due to arterial insufficiency ).. color doppler test is for confirm diagnosis.. this is reduced blood supply to the affected part.. ie dhamani gata vaata ( due to dhamanipraticchay). In reference to paandu acharya charak mentions shishir dvesha ( cold intolerance) , in paandu shishir dvesh is present in both upper n lower limbs , but in patient of PAD only in lower limbs(LL); means alpa rakta in LL ie due to arterial insufficiency/PAD/Atherosclerosis. Examine in this manner too.. t/t ekangaveera rasa , arogyavardhini vati , combination of dashamoola, pushkaramoola , erandamoola , raasnaa , punarnavaa , devadaaru , daruharidra , kushtha , amritaa , vidanga , chitraka , vachaa , trikatu ,darbha.. shilaajita.. bhallaataka kalpa if tolerable..

20 Oct 2012

Acharya chakrapani on ch.ni.7/5;buddhi vibhramattu nityam anityam, priyam cha apriyam iti pashyati; vachanam hi-visham abhinivesho yo nitya anitye hita ahite. gyeyah sa budhivibhranshah,samam budhirhi pashyati. ch.sh.1/99). in modern health sciences it is obsessional compulsive disorder(OCD) and in ayurved atattvabhinivesh...

3 April 2015

**Neurocutaneous syndromes ( phakomatoses)** ; neurofibromatosis type 1 is an autosomal dominant disorder ( sahaja) . The NF1 gene on chromosome 17q11.2 encodes protein , neurofibromine , GTPase activating protein ( components of maansa dhaatu). Mutations of the NF1 gene result in a large number of nervous system tumors including neurofibromas, plexiform neurofibromas , optic nerve gliomas , astrocytomas , and meningiomas.neurofibromas appear as multiple , soft , rubbery cutaneous tumors.. The involved structures are sthaana of vaata karma. As per Ashraya-ashrayi bhaava involvement of vaata is responsible to

develop utsedha/granthi/tumors in vaata sthaana . Multiple, Soft and rubbery presentation of cutaneous tumors also indicate the involvement of vitiated vaata and maansa as dooshya.. involved proteins are component of maansa dhaatu , tvaka is upadhaatu of maansa dhaatu so cutaneous tumors are manifested. Vaata as dosha and tvaka, maansa are as dooshya.. tumors are sanga pradhaana vyaadhi with imbalance between stimulators (↑ vaata ) and inhibitors ( ↓ kapha) of cell proliferation. There is a controlled rate of cell division due to presence of praakrita aavarana to restrict the increased proliferation rate. Tumorous growth occurs due to loss of praakrita aavarana which results in either increased stimulators or decreased inhibitors or both leading to sanga initially followed by atipravriti.. the choice of drugs include to suppress stimulators and /or activate inhibitors.. taamra vachaa chitraka like drugs penetrate sanga and will reach in inside of cells ( intracellular action ) , kaanchanaara trifala haridra kushtha bilva mustaa vidanga like drugs may act as lekhaniya drugs.. amrita haritaki aamalaki darvi tulasi haridra like drugs may act as controllers of cell division and on mutation of gene.. Its not curable but can be thought in this direction to search best remedies..

### **Hypertrophy Of Bone - Hyperostosis ; अध्यस्थि - अस्थि प्रदोषज विकार .**

Bone increases in substance in two ways, which are not essentially different from one another.

1. In the one case, while the density of the bone remains unchanged, new osseous substance is deposited on its surface, beneath the periosteum, and augments it in breadth and thickness. The size of the medullary canal remains the same, but the compact substance around it is thicker than before (External hyperostosis).

2. In the other case, the increase of substance is internal, proceeding from the Haversian canals, and in the end from the whole medullary system. The bone becomes more dense, not only in its compact layers, but also in its cancellous part; the walls of its cells, and the bony threads of which its network is composed, increase in thickness; and, by a kind of concentric hypertrophy, as it were, the medullary cavity diminishes in size, and the diploe disappears. We may call this state an internal hyperostosis; it constitutes also the induration (sclerosis) of bony tissue.

The two forms very commonly occur together, and thus in a twofold manner augment both the bulk and the weight of the bone.

25 Sept 2012

Acharya charak mentioned astheeni durbalaani (osteomalacia) laghooni (osteopenia) cha, features of asthikshaya.

Read more: 30 Jun 2015

Vit D deficiency in ayurveda ; shramah sandhishaitilyam in asthi kshaya . Astheeni durbalaani laghooni ch in majjaa kshaya.. in vit D deficiency asthi and

majjaa both kshaya occurs.. as per ayurveda grahani dosha, asthi majjaa kshaya are to be considered for deciding treatment.. daadimaadi ghrita/shatavari ghrita, pancha tikta ghrit guggula, godanti , laghumalini vasant , madhumaluni vasant kukkutaandaka tvaka bhasma , amrita amalaki musta arjun padmyakashtha kamal ashwagandha shunthi are found effective clinically n biochemically too in vit D deficiency...

23 March 2015

**Salient features of amlapitta** ; The acid environment within the stomach leads to cleavage of the inactive precursor to pepsin and provides the low pH (<2) required for pepsin activity. Pepsin activity is significantly diminished at a pH of 4 and irreversibly inactivated and denatured at a pH of >7. Achlorhydria leads to hypergastrinaemia. Enterochromaffin- like (ECL) cell hyperplasia with frank development of gastric carcinoid tumors may result from gastrin trophic effects. Hypergastrinaemia and achlorhydria may also be seen in non pernicious anaemia-associated type A gastritis. Drugs containing katu rasa maintain required pH to activate pepsin for its action on protein digestion..therefore its known as deepaniya .. Proton pump inhibitors can develop mild to moderate hypergastrinaemia and in turn carcinoid tumors in some experimental animals.. ayurveda drugs effective in amlapitta never develop such side effects , only because of katu and tikta rasa.. katu rasa produces achchha pitta not vidagdha pitta... Heart burn and regurgitation are common symptoms of GERD (urdhvaga amlapitta). Inherent in pathophysiologic model of GERD is that gastric juice is harmful to esophageal epithelium. However , gastric acid hypersecretion is usually not a dominant factor in development of esophagitis. One caveat is with chronic H.pylori gastritis, which may have a protective effect by inducing atrophic gastritis with concomitant hypoacidity. Pepsin, bile and pancreatic enzymes within gastric secretion can also injure esophageal epithelium. Bile warrants attention because it persists in refluxate despite acid suppressing medications. Bile can transverse



cell membrane , imparting severe cellular injury in a weakly acidic environment, and has also been invoked as a cofactor in pathogenesis of Barrett's metaplasia and adenocarcinoma. Hence, the causticity of gastric refluxate extends beyond hydrochloric acid.. kutaki , amrita , kiraatatikta , shataavari , aamalaki ,avipattikara churna , bhoonimbaadi kvaatha, daadimaadi ghrita , kushmaanda avaleha ,etc drugs are best options in urdhvaga amlapitta and to prevent metaplasia...

13 Jan 2015

Barrett's mucosa.. avipattikar churna or vati (+ yashtimadhu aamalaki amrita shatavari haritaki), kamadudha vati , laghusootasekhar rasa and bhoonimbaadi kwatha..shatavari or daadimaadi ghrita..

Gastroesophageal reflux - esophageal inflammation - esophageal ulcer - esophageal stricture - barrott 's mucosa - adenocarcinoma of esophagus.. urdhvaga amlapitta - pitta- daarana, dahana, jaarana of cells, kaphanubandha - sthairy- fibrosis - sanga - change in nature of cells - arbuda.. Barrott 's mucosa may be site of Ca .. Tamra kalpa , lekhanaya dravya , jeevaniya dravya and pittakaphaghna are choice of treatment. No liquid one hr after dinner , early sleeping n early awakening.. try to convince patient not to take heavy meals , spicy diets cold beverages alcohol tea coffee smoking.. pomegranates are most useful fruit.. aamalaki is to prevent Ca.. and haritaki ,nishottar, kutaki help as prokinetic drugs. Shatavari, amrita ,amalaki ,haritaki, yashtimadhu as rasayan . Kamadudha laghusootashekhar bhunimbaadi daadimaadi ghrita as dosha n vyadhipratyanika

17 August 2014

**Gulma** is vaata vyadhi due to vyaahata gati of vaata.. Here partial obstruction in koshtha leads to resistance to vaata gati .. e.g. Vaatika gulma : sthaana vikalpa sanshthaana vikalpa rujaa viklapa indicate vaishamya gati of vaata , vidavaata sangam indicates obstruction to passage of vaata . Shishir jvara indicates presence of infective cause (helicobacter pylori ) , karoti jirne abhi adhikam prakopam bhukte mridutvam... ( duodenal pathology ) ( ch.chi 5/10-11).. we can correlate

with duodenal ulcer . T/t deepaniya ghrita like daadimadi ghrita , Avipattikara churna , combination of aamalaki shataavari yashtimadhu amrita and jataamaansi , pravaala panchaamrita/kamadudhaa , laghu sootashekhara rasa , bhoonimbaadi kvaatha etc...

1 Dec 2014

Ayurveda perspective of celiac disease ; gluten, is present in wheat barley and rye, is protein so it causes kaph prakopa and later turn in triggering of vitiation of vaata in genetically susceptible persons.. sthaana sanshraya occurs at intestine and leads to grahani dosha.. sequence of samprapti gluten + genetical predisposition kapha prakopa + vaata parakopa autoimmunity inflammation destruction of intestinal villi grahani dosha impairment in paachana/saara kitta vibhajana/absorption (impaired agni and samaana vaata ) maldigestion and malabsorption sankshepatah kriyayogo nidaana parivarjanam , deepaniya paachaniya drugs/ kashaaya , takraarishta , parpati kalpa..eg.

Sanjivani vati , rasa parpati, combination of balachaturbhadra + hingavashtak churna + bilva + musta + manjishtha ..

28 Nov 2014

Trapa bispinosa( singhara,paniphal) ; fruits contain iron multivitamins Ca Na K Mn Zn phosphorus citric acid saponins phenols alkaloids flavonoids glycosides steroids etc. Contents of fruits act as analgesic spasmolytic anticholinergic anaesthetic aphrodisiac astringent antipyretic antidiarrheal appetizer diuretic hemostatic immunomodulator neuroprotective antibacterial antiplatelets aggregator. Its indicated in diarrhea dysentery ophthalmopathy ulcers wounds hemorrhage hemoptysis fever leprosy fracture lumbago pharyngitis bronchitis leucorhea threatened abortion impotence menorhagia fatigue general debility etc.. its good source of healthy nutrients.. commonly used by indians in fasting.. Its also indicated in UTI , Thrombosis , neurological disorders , autoimmune diseases..Being anticholinergic its indicated in bradycardia..it is best in gastritis

due to pitta shaamaka and anticholinergic action.. vagus mediated hydrochloric acid secretion is inhibited by singhara..

10 Nov 2014

Gastritis-ayurveda perspective; Inflammation of gastric mucosa is due to backward influx of H ions into gastric mucosal cells caused by aspirin alcohol H.pylori spices stress etc.. local irritating causes or decreased blood supply to mucosal cells as in burn or due to impaired mucosal resistance.. in all these causes pitta is only component to play a role in genesis of gastritis.. due to decreased prostaglandin synthesis there is decreased mucus secretion as well sodium bicarbonate secretion.. they are component of kapha , it means there is decreased kledaka kapha which vitiates vaata and promote backward flow of H ions which in turn increases sthaanika pitta and Ph in cells are increased leading to erosion/ulceration of gastric mucosal cells.. Yashtimadhu amritaa aamalaki kiratatikta are perfect drugs for pitta shaman... jataamaansi is best in stress induced gastritis.. avipattikara, Haritaki are best for prokinetic purpose ( anulomana).. daadimaadi ghrita / shataavari ghrita / drakshaa ghrita are best for pitta and vaata.. yashtimadhu aamalaki like drugs increase mucus secretion.. Amritaa like drugs enhances prostaglandin synthesis to increase mucus secretion and sodium bicarbonate secretion... annadrava shoola, parinaama shoola , annavrita vaata , vaataja gulma are some examples where stomach and duodenum are site of diseases..

30 March 2016

### **My Approach to IBS in ayurveda perspective**

Gastrocolic & colorectal transit time is affected in IBS.. Shorter transit time due to hypermotility leads to IBS-D , and in IBS-C longer transit time is present.. IBS can be correlated with vaataja grahani.. In IBS-D, apaana avrit vyaana and in IBS- C vyaana aavrita apaana , in IBS-M samaana aavrita apaana should be considered. Line of treatment is specific for various subtype of IBS.It may be concluded that aggravated vāta has following effect

- 1) Impairs contraction and relaxation of sphincters thereby impairing the entry and exit of food within GI tract.
- 2) Either increases peristalsis thereby reducing transit time and thereby impairing the digestion or may reduce the peristalsis (due to kaṣāya rasa) and increase the transit time thereby hampering digestion.
- 3) Vāta can cause atrophy by rukṣa, khara guna and thereby reduce secretion of digestive glands and enterendocrine hormones.
- 4) Vāta can present neural transport of specific ions, amino acids and thus impair the digestion.All the above process proves aggravated vāyu encompassing the agni and leading to indigestion. Gut - Brain relationship has been observed by research; Praana - samaana - apaana interrelationship is important to digestion and absorption of nutrients and vitamins..Spastic colon is very common in elders , and due to autonomic nervous system dysfunction as in DM ; purishaavrita vaata , udaavarta , pakvaashayagata vaata and guda gata vaata are concerned with slow transit time.. In all these conditions snigdha anulomana is reffered , for which eranda sneha , or gandharva haritaki , or/and sinhanaada guggula should be given for effective treatment.. Fibres rich diet , good amount of water , and proper exercise schedule help a lot in normalizing colon motility.. Bilva , karkatashringi , ativishaa , manjistha , hingaavashtaka like drugs are more



effective in IBS-D , avipattikara is better choice in IBS-C , in IBS- M combination of above mentioned drugs (as per conditions and as per consultant ) should be given.. Bhaanga containing kalpa is not choice of drugs to use for long term since IBS relapse frequently. Addiction is fear. Praana - udaana interplay in addiction and hamper feedback mechanism..

25 Feb 2014

### **Diarrhea**

Diarrhea is defined as passage of abnormally liquid or unformed stools at an increased frequency. Diarrhea is classified as acute if <2 weeks, persistent if 2-4 weeks , and chronic if > 4 weeks in duration.. pseudodiarrhea of IBS or proctitis and fecal in continence due to neuromuscular disorders or structural anorectal problems must be differentiated.. diarrhoea and urgency , especially if severe , may aggravate or cause incontinence. Acute diarrhea is mostly infectious but also noninfectious in origin. Appropriate antibiotics in infective and ant motility and antisecretory agents such as loperamide can be useful in noninfective diarrhea.. Treatment of chronic diarrhea depends on the specific cause. For examples; elimination of dietary lactose for lactase deficiency or gluten for celiac sprue , use of glucocorticoids or other anti-inflammatory agents for IBDs , omeprazole in Zollinger Ellison syndrome , cholestyramine for ileal bile acid malabsorption , octreotide for malignant carcinoid syndrome , indomethacin for medullary carcinoma of the thyroid , pancreatic enzyme replacement in pancreatic insufficiency , tetracycline and folic acid in tropical sprue , clonidine ( alpha2-adrenergic agonist) in diabetic diarrhea. For all patients of all types of diarrhea, fluid and electrolytes repletion is an important component of management.. Replacement of fat soluble vitamins in chronic steatorrhea ( fat in stool )..

18 March 2016

Zinc supplementation in the management of diarrhoea.

Biological, behavioural and contextual rationale ;

A continuing lack of safe water and adequate sanitation in many parts of the world means that diarrhoea remains the leading cause of death among infants and young children in low- and middle-income countries . Every year more than a million children under five years of age succumb to the fluid loss and dehydration associated with the majority of diarrhoea related deaths. It is estimated that 13% of all years lost due to ill-health, disability, or early death (so-called “disability-adjusted life years”) are caused by diarrhoea. Good guidelines on the clinical management of diarrhoea among the world’s most vulnerable children therefore remain critical. There are two simple and effective treatments for the clinical management of acute diarrhoea: use of low concentration oral rehydration salts (ORS) routine use of zinc supplementation, at a dosage of 20 milligrams per day for children older than six months or 10 mg per day in those younger than six months, for 10–14 days. Oral rehydration is a well-known and relatively simple treatment approach . Zinc supplementation has been found to reduce the duration and severity of diarrhoeal episodes and likelihood of subsequent infections for 2–3 months. Zinc supplements are generally accepted by both children and caregivers and are effective regardless of the type of common zinc salt used (zinc sulphate, zinc acetate or zinc gluconate) . Supplementary zinc benefits children with diarrhoea because it is a vital micronutrient essential for protein synthesis, cell growth and differentiation, immune function, and intestinal transport of water and electrolytes . Zinc is also important for normal growth and development of children both with and without diarrhoea . Zinc deficiency is associated with an increased risk of gastrointestinal infections, adverse effects on the structure and function of the gastrointestinal tract, and impaired immune function . Dietary deficiency of zinc is especially common in low-income countries because of a low dietary intake of zinc-rich foods (mainly foods of animal origin) or inadequate absorption caused by its binding to dietary fibre and phytates often

found in cereals, nuts and legumes. Although the benefits of zinc supplementation in the management of diarrhoea have been established, there remain a number of barriers to the widespread implementation of this treatment strategy. Currently, zinc is not used to treat most cases of diarrhoea because the known benefits of zinc supplementation are still not widely appreciated by physicians and health-care workers in developing countries . There is a need to establish the optimal dosage and to investigate whether the same benefits of zinc supplementation are also applicable to children in middle- or high-income nations . There is also concern that high zinc intakes may compete for absorption with other micronutrients such as iron and calcium. This, in turn, can have unintended negative consequences for children's health and development. Studies are needed to help identify subpopulations that would benefit most in resource-limited settings and to ensure access to zinc supplementation, especially for those families whose children are most at risk of diarrhoea but may not be able to afford treatments that include zinc supplements. However, zinc deficiency remains difficult to diagnose because measuring serum zinc levels is not necessarily accurate for this purpose .

Currently, only a very small proportion of children in need have access to zinc supplementation. Guidelines on the use of zinc supplementation in the management of diarrhoea may accelerate progress towards the United Nations Millennium Development Goal 4 for reducing child mortality by two-thirds by 2015

23 June 2014

**Achalasia** is caused by loss of ganglion cells within esophageal myenteric plexus.. excitatory ( cholinergic) ganglionic neurons are variably affected and inhibitory (nitric oxide) ganglionic neurons are necessarily involved. Functionally inhibitory neurons mediate deglutitive LES relaxation and the sequential propagation of



peristalsis. Their absence leads to impaired deglutitive LES relaxation and absent peristalsis.. this is auto immune process attributable to a latent infection with human HSV 1 combined with genetic susceptibility.. C/F : dysphagia , regurgitation , chest pain and weight loss.. therapy is directed at reducing LES pressures so that gravity and esophageal pressurization promote esophageal emptying . Nitrates or calcium channel blockers are administered before eating , advising caution because of their effects on blood pressure..The only durable therapies are pneumatic dilatation and Heller myotomy...Ayurved ; impaired deglutitive LES relaxation is due to vitiated vyaan and decreased peristalsis due to aavrit udaan ( prayatna, urja ,bala karma) so it may be vyaanaavrita udaan .. as immunity ( bala) is impaired so udaana karma is being decreased... anulomana and laghu bhojan.. drugs like avipattikar , haritaki , amrita , shatavari , pushkarmool etc are effective in somewhat extent..Patients with advanced achalasia are at risk for brochitis, pneumonia, or lung abscess from chronic regurgitation and aspiration.Acharya charak mentions vidradhi as one of upadrava of aavarana..( aavritaanaam upekshanaat ch.chi 28/236-237..

4 May 2013

Hepatic encephalopathy ; Tandra (drowsiness), Moh(confusion), Nasht Sangya(coma) are present in kumbh kamala...ch.ch.16/38-39

25 Sept 2012

Acharya Charak mentioned kuksheratimaatravidhih(distension of abdomen with bulged flanks),siraantardhanagamanam(dilated veins on abdomen,caput medusae), udakpoornadritisankshobh(fluid thrill) udakpoornadritisansparshatvam cha(shifting dullness) in jalodar ch.chi.13/48.

Acharya Charak mentioned mootrasang as complication of jalodar (ch.chi.13/49).here mootrasang is due to hepatorenal syndrome, a complication of cirrhosis of liver. In cirrhosis of liver, ascites & splenomegaly are common features due to portal hypertension.



15 May 2014

**Omphalolith:**

Because of poor hygiene in deep seated and retracted umbilicus , especially in obese person , there is accumulation of sebum and keratin leading to stone formation known as omphalolith , later may be ulcerated and infected at time of diagnosis.. its resembling to a malignant melanoma and differntial diagnosis with umbilical cholesteatoma..(an accumulation of crumbling, fetid masses in the umbilicus, often times accompanied by seborrhoea which may lead to abscess formation) omphalolith is benign tumor and need surgical removal.. in ayurved perspective.. its like a pitica in nabhi , similar to ashmari , due to avarodh in svedavah srotus, there is sang of components of swed leading to formation of hard , smooth , black bolus.... its simply sang of dosh.. such mass also may be malignant melanoma ; because of the variations in vascularity and residual embryonal connections of the umbilicus with the peritoneum and the other intraabdominal organs it may be present due to peritoneal or colon cancer.. its due to spreading of cancer from colon to umbilicus.. need to do biopsy to confirm diagnosis.. in ayurved its karkata arbud.. treatment is surgical removal, drugs acting on meda, mamsa & twacha especially lekhanii dravya with twachya may be more effective. Hygiene of umbilicus should be checked at regular interval to avoid any accumulation. Application of oil in umbilicus before bath is practiced in India since centuries. Take care of each & every part of the body to assure complete healthy life.

## **Approach to infertility in men ;**

### **DIAGNOSTIC EVALUATION:**

The couple should be evaluated together to determine whether the problem resides in the male partner, the female partner, or both. The objectives of evaluation are to exclude treatable conditions--gonadotropin deficiency, obstruction, and coital disorders--and identify those who are candidates for assisted reproductive technologies, those who are sterile and should consider adoption or artificial insemination using donor sperm, and those who should undergo genetic screening. All infertile men should undergo several semen analyses according to the World Health Organization manual, as well as measurements of testosterone, LH, and FSH levels. Hormone measurements can help determine whether the patient has gonadotropin deficiency (low testosterone and low or inappropriately normal LH and FSH), primary testicular failure (low testosterone, elevated LH and FSH), spermatogenic failure (normal testosterone and LH, elevated FSH), or androgen resistance (high testosterone, elevated LH). A majority of infertile men have normal testosterone, LH, and FSH levels. Obstruction should be ruled out in azoospermic men with normal testosterone, LH, and FSH levels.

### **GENETICS:**

Yq microdeletions are the most prevalent cause of spermatogenic failure in men with azoospermia or severe oligozoospermia. Infertile men with azoospermia or severe oligozoospermia should undergo karyotyping and testing for Yq microdeletions. Men with congenital absence of vas should be tested for cystic fibrosis transmembrane conductance regulator mutations.

### **THERAPY:**

Gonadotropin therapy is highly effective in gonadotropin-deficient men. Intracytoplasmic sperm injection (ICSI) has emerged as the treatment of choice

for idiopathic male factor infertility. However, ICSI is expensive and associated with a higher risk of multiple gestation, low birth weight, preterm delivery, perinatal complications, and chromosome aneuploidy than naturally conceived pregnancies. Men considering ICSI should be offered karyotyping, Yq microdeletion testing, and genetic counseling by counselors experienced in reproductive disorders.

anatomical or functional correlation of subtypes of vāta is attempted here for a rough and overall understanding for beginners. Prāna Vāyu is concerned with consciousness, arousal, heartbeat, vomiting, breathing, cough, hiccup etc. The modern functional analogue may be compared with brain stem and reticular formation which directly control cardiovascular / respiratory systems, pain sensitivity, alertness, awareness, and consciousness. Udāna is concerned with language, learning, mood, initiation, judgment, intellect, recall information etc. The prefrontal cortex, sub cortical areas and parts of limbic system along with association areas may be understood as functional areas of Udāna. Vyāna is concerned with control of skeletal muscle activities, control of hemodynamics, sweating etc. Post-lateral and dorso-medial hypothalamus - sympathetic stimulator, primary motor area, basal ganglia, extra pyramidal tract and autonomous nervous system are part and parcel of vyāna vāta. Samāna and Apāna can be considered together. Gastro Intestinal Tract based enteric nervous system (2nd brain), (brain- gut axis - more than 100 million neurons), celiac plexus, sacral plexus etc may be analogue for apāna and samāna.

## **An Ayurvedic Approach to Cardiology**

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Haraterdadaterete hriday shabdah (shatpath bramhan)..means: hri-aharan - venous return..d-danarthak- stroke volume..y-gatyarthak-contractility of myocardium and heart rate..

Heart disease is now the world's leading causes of death, claiming 17.3 million lives each year. Latest statistics in India suggest that, there are roughly 30 million heart patients. Of the 30 million heart patients in India, 14 million reside in urban areas and 16 million in rural areas. The Indian rural population and urban poor especially are facing a "double burden" - with incidences of acute diseases continuing, while there is a rapid growth in incidences of chronic diseases, "If the current trend continues, by the year 2020, the burden of atherothrombotic cardiovascular diseases in India will surpass that of any other country in the world."

Currently, the key challenges that face cardiac care in India are inadequate facilities, accessibility, the price tag attached to efficient and effective treatment, lack of awareness of non-communicable diseases. The growth of heart diseases is dependent on a number of interlinked factors such as ageing, changing lifestyles, bad eating habits and rapidly evolving socio-economic determinants like access to healthcare.

Ayurveda holds the breakthrough to the key challenges that the India and in turn the world is facing in present era in cardiac care. Various scattered literature can



be brought together and a clinical cardiology in Ayurveda can be given to modern society.

The word HRIDAYA is made up of HRI- DA- YA. HRI means Aharan, physiologically it means Venous Return or Preload, DA means Danarthaka or the Stroke Volume i.e. Afterload and YA means Gatyarthaka i.e. Rhythmic Contractility of Myocardium or heart rate.

Heart is a aerobic organ and seat for oja, indriya, sadhak pitta, atma, pranvayu, avalambak kapha, mana and vyanvayu. It is mulasthan for pranvaha and rasavahasrotas and due to rasa vikshepana karma it gets associated with udakvaha, raktavaha and all the srotas of the body.

Charak in Rasayan adhyaya has describe gramya aahar which is the key for the increase of non communicable disease. It consists of Amla, lavana, katu, kshar dominated diet which is cause for vitiation and abnormal increase of rakta dhatu. Shuska shaka, shuska mansa, oil extracted paste of sesame etc. and various consumables having ruksha quality have a very low nutritional value. Klinna, guru, pishtanna, adhyasana, sedentary lifestyle and abhishyandi aahar which are source of extra calories. Virudha, asatmya, visham aahar, and paryushit anna which have an impact on our immune system. Day sleep, Indulging in excessive exercise and sex, Alcohol intake, which increases oxidation process and release oxidants and free radicals and Pshycological factors such as bhaya, krodha, lobha, shoka, moha which are cause for the neuro-hormonal imbalance, for example excess adrenaline secretion and in turn over sympathetic activity.

Indulgence in gramya aahar results as laxity in Mansa, Vidahyate (inflammatory changes) in rakta, excessive and abnormal meda and Oja kshaya. Sama meda is enriched in prithvi and aap mahabhuta caused due to altered ratio between dravatva and ghanatva resulting into increase Pratighata and decreased apratighata. Resistance to Vyan vata results into increased rheological forces within the vessels and chambers of heart. Increase rheological forces cause injury

to the srotas thereby causing proliferation of new vessels. But sama meda due to its quality to adhere to vessel wall further increase pratighat continuing the vicious cycle of injury to srotas (vessel) and proliferation of new anusrotas (vessels). This contributes to dhamanipratichaya a nanatmaja vyadhi of kapha dosha. Concept of Madhumeha as explained in Ca. Su. 17th chapter and pathogenesis of Urustambha as explained in Ca. Ci. 27th chapter is necessary to understand the pathogenesis of atherosclerosis in ayurveda. Therefore due to srotoavarodh disparity origins between the nutritional demand of the cardiac muscles and nutritional supply through the vessels which have undergone dhamanipratichaya leading to hritshula (angina) and vatic hridroga (IHD).

Angina pectoris, commonly known as angina, is severe chest pain due to ischemia (a lack of blood and hence oxygen supply) of heart muscle, generally due to obstruction or spasm of the coronary arteries. Sushruta in uttartantra gulma chikitsa adhyaya has mentioned about hritshula Kapha, pitta or both when impedes the gati of vata which is associated with rasa especially in cardiac muscles leads to chest pain (hritshoola/ angina).

Psychological factors like krodha which vitiates pitta and bhaya, shoka which leads to pran vayu dusti leading to sympathetic overactivity and inturn influencing action of vyan vayu in a Hina satva person (Type A personality) leading to increase in heart rate. If this takes place in already compromised cardiovascular disease patient it leads to symptoms like angina and vatic hridroga.

Anatomical changes in heart like, aortic stenosis obstructs the left ventricular outflow of blood reducing the cardiac output leading to increased contractility of left ventricular muscles later on converting into Left Ventricular Hypertrophy. If demand of nutrition for the increased surface area, if not supplied leads to angina and later on precipitate as hridayagata vata.

Vata is necessary for the coordinated functioning of heart. If vata gets vitiated the conduction defects may occur. Tachycardia and bradycardia are the 2 main

classification of impaired cardiac conduction. Dara or dardarika as explained by Chakrapani or hridrava as explained by Yogindranath Sen explained the tachycardia. Various rhythmic and arrhythmic tachycardias have been described by Modern science for eg. Sinus tachycardia, Atrial fibrillation, Atrial flutter, AV nodal reentrant tachycardia, Accessory pathway mediated tachycardia, Atrial tachycardia, Multifocal atrial tachycardia, Junctional tachycardia, Ventricular tachycardia, supraventricular tachycardia etc.

Hridstambha explains the bradycardia. Stoppage, obstruction, suppression are various meaning of stambha. Sinus bradycardia, Sick sinus syndrome, AV block etc explain the condition of slow heart rate. Asystole, also known as flatline, is a state of no electrical activity from the heart and therefore no blood flow. It results in cardiac arrest. It may also be noted that tachycardia may later convert into asystole. Thus various conditions resulting into tachycardia and bradycardia may be considered in vataj hridroga.

Pittaj hridroga includes infective conditions of cardiac diseases like infective endocarditis/ myocarditis/ pericarditis, Rheumatic heart disease, infective cardiomyopathy. Alcohol, chemotherapeutic drugs, heavy metals like arsenic etc induced heart disease has similarity with pittaj hridroga.

Stabdha, Sputa and Stimita are the 3 lakshana mentioned by Caraka for kaphaja hridroga. The Sanskrit meaning of the word stabdha is firmly fixed, stiff, rigid, immovable, paralyzed, senseless, dull, solidified, tardy, slack, slow whereas sputa means insensible, dull, resting, latent, inactive and Stimita means Fixed, rigid, unmoved, motionless, steady; paralysed, flowing gently along. It means heart is motionless, inactive, slow, solidified, rigid, these 3 words extent the scope of kaphaja hridroga from non infective cardiomyopathy to cardiac tamponade.

Pathogenesis of various Cardiac diseases can be understood through scattered references in Samhita after going through chapters related to sthauilya, raktapitta, gulma, rajyakshma, kshatakshinna, shoth, grahani, hikka shwas, kasa,



trimarmeeya adhyaya, urustambha, vatarakta and especially avaran from vatavyadhi.

Primary approach to the prevention and treatment of cardiac disease can be understood from the dincharya and ritucharya adhyaya. Importance of early morning exercise, prudent diet, lifestyle, avoiding smoking and tobacco products, following aachar rasayan, meditation and good management of stress are the basic line of treatment for all the non communicable disease.

Prudent diet consisting of varied eating pattern should be followed. Preference should be given to fish, chicken, low fat dairy products. Consume more unrefined carbohydrates such as grains product. Salt & alcohol intake should be moderate and diet should consist of antioxidants like amalaki, lemons, spinach, turnip leaves, watermelon, sweet potatoes, carrots, tomatoes, pumpkin, wheat grass juice, oranges, guava, mung etc. Various fibrous diet, vegetables, seasonal fruits, omega 3 fatty acids should be taken.

A multiple direction approach should be kept in treatment of Cardio-Vascular Disease. Causative factors and the pathology should be understood than only treatment should be initiated. Role of sama meda, rasa-raktavaha and pranvaha srotas, vata dosha, grathita rakta, hridaya kriya, rogatkarshana, psychological status all these factors should be considered and then multidrug combination should be decided.

Research has shown that various herbal drugs have multiple actions which can be useful in cardiology. Drugs like Kamalkshar, Darbha, Kusta, Paravatashakrit, Mrinal, Palash kshar, Priyangu kshar act on grathit rakta (thrombus). Drugs like Maricha, Chitrak, Daruharidra, Rason, Tulsi and Vacha act on sama meda. Amalaki, Haritaki, Punarnava, Shatavari, Shalparni, Sariva, Manjista act on rasa and raktavahasrotas. Vasa , Amrita, Punarnava, Amalaki, Pushkarmoola, Kusta, Kachora, Kantakari and Brihati have action on pranvahasrotas. Some Drugs like Punarnava, Gokshur,



Musta, Ushir, Dashmula, Varun have mutrala effect so reduce the hypervolaemia. Drugs like Arjun, Brahmi, Tulsi, Guggulu and Punarnava have hridya effect.

Role of Shodhan karma in hridroga has its own limitation. It should be ordered considering the basic parameters, cardiac preload, afterload, age etc. If preferred mridu prayog should be carried out that too understanding the bala of patient and dosa. Yapana Basti can be reassured.

A brief approach has been mentioned above but the sutras mentioned by our Acharya have the capacity to explain complete Cardiology in Ayurveda.

Acharya charak mentions bhavati ashmavritam yatha in kaphaj hridroga.ch.su.17/35.The echocardiogram shows thickened & calcified pericardium in constrictive pericarditis..

16 April 2013

Paradoxical pulse, the important clue to the presence of cardiac tamponade(massive pericardial effusion) consists of a greater than normal (10 mmHg) inspiratory decline in systolic arterial pressure. When severe,it may be detected by palpating weakness or disappearance of the arterial pulse during inspiration...

22 May 2013

Dushayitva rasam dosha vigunaah hridayam gataah. Kurvanti hridaye baadhaam hridrogam tam prachakshate. Su.u.t.43. Heart failure is a clinical syndrome in which an abnormality of cardiac structure or function is responsible for the inability of heart to eject or fill with blood at a rate commensurate with the requirements of the metabolizing tissues..

1 April 2015

### **HCM and HOCM with ayurveda perspective**

Hypertrophic cardiomyopathy is characterized by non dilated left ventricular hypertrophy. Later there may be hypertrophic obstructive cardiomyopathy due to left ventricular outflow tract pressure gradient. Three basic mechanisms are involved ; (1) increased left ventricular contractility ( effect of prakupita kapha ), (2)- decreased ventricular volume (preload),( aadaana karma of praana vaayu is impaired ), (3)- decreased aortic impedance and pressure (afterload) -( vyaana vaata karma haani ) 2&3 are because of hridaya gata vaata ( angina pectoris , fatigue and syncope are present with dyspnea , a common symptom due to diastolic dysfunction/ impaired left ventricular filling indicate predominance of vaata Kaphaja hridroga with hridaya gat vaata can be considered in HCM and HOCM.. Hridayarnava rasa , chandraprabha vati , gokshuraadi guggula , kvaatha of combination of dashamoola arjuna punarnava gokshuru varuna haritaki devadaru pushkaramoola kamal shatavari ashwagandha chitraka vacha shunthi . Shaalaparni with milk. Kamadudha , arjunaarishta , hrida basti by dashamoolaadi tail or prepared as per ch.chi 26 vaatika hridroga..Advice for salt restriction , no physical and mental stress , no alcohol, no smoking , small bolus of food at a time 4-5 times per day. Pomegranate grape orange fig are good fruits .. Gandharva haritaki can be added at night for proper bowel motion to avoid straining during defaecation...Food intake increases blood suply to splanchnic circulation so demand is increased which in turn develops overload on failing ( compromised ) heart and manifests dyspnea fatigue giddiness chest pain... Its due to vaata prakopa in hridayashtha sthaan ie hridaya gata vaata. Anshumati sapayasaa is indicated by acharya charak...

I have elaborated 3 specific underlying pathology in HCM and HOCM with vaata predominance , but initial factor is kapha prakopa leading to non dilated

cardiomyopathy , increased mass of myocardium , mass represents sthoola bhaava so its due to kapha , later contractility preload and afterloads are impaired . due to diastolic dysfunction dyspnea manifests , due to increased mass demand for O<sub>2</sub> is increased and that is not supplied adequately so there is imbalance between demand and supply leading to ischaemia and chest pain ie due to apatarpana vaata prakopa in hridaya so hridaya gata vaata can be considered.. decreased cardiac output (due to left ventricular outflow obstruction - sanga ) causes decreased effective arteriolar volume and renal hypoperfusion resulting in activated renin angiotensin aldosterone system to manifest salt and water retention vasoconstriction and remodelling of ventricle. These are consequences of disease process and can be considered as vitiated vaata to bring ambu dhatu to retain beneath tvaka and maansa ie edema.. treatment plan includes to work on 3 basics ; arjuna shatavari kakamachi ashwagandha for strength of contractility to maintain diastolic function. Punarnava gokshuru varuna to reduce preload and pushkaramoola dashamoola to maintain normal after load.. chitraka vacha trikatu to decrease sanga or obstruction.. hridayarnava rasa contains tamra and kaakamaachi so better option in this condition.. chandraprabhavati gokshuradi guggula to decrease congestion , gokshuru is hridya too. Kamadudha acts on action potential to provide normal ionic changes across cell membrane . Angiotensin 2 is potent vasoconstrictor( increases peripheral resistance) , increases sympathetic activity (increased cardiac output), secretion of arginine vassopressin so increase renal reabsorption of water , increase aldosterone secretion so promote salt and water retention( increase venous return so increased cardiac output ) →hypertension.. salt is major cause of activated RAAS - pitta prakopaand rakta vridhhi. Kapha prakopa and dhamanipratichaya...HTN. Aamaashaya gata vaata manifests shvaasa , as pranavaha sroto dushti since pitta sthaana samudbhava is mentioned in shvaasa roga.. here shvaasa is one of symptom of HCM.

Hridaya as a whole organ is moola sthaana of prana vaha rasa vaha and rakta vaha.In dhamanipratichaya sakapha meda and maansa interplay to develop

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uplepana on dhamani .. it leads to avarodha of vaata gati.. it should not be considered as dhamanipoorana: In Aortic stenosis pathogenesis occurs similar to as that of atherosclerosis so modern dr advice statins in AS . CAD, AS and HCM are different disease entities .. but in AS and HCM angina pectoris is present without CAD , only due to increased demand caused by hypertrophy..

5 Aug 2014

Ventricular premature complexes ( VPCs ) are common and increase with age and the presence of structural heart disease.. EKG is helpful for diagnosis, there may be patterns of bigeminy or trigeminy or multiformed.. Prabhakar vati 250 mg /day , combination of arjun , shatavari , brahmi , kamal in equal quantity ; 6 gm / day are found effective to eliminate symptoms associated with palpitation.. On 15 days treatment EKG revealed no PVC....

### **Preventive cardiology; certain salient features**

1. It's thought that most cases occur when something affects the heart's development during about week five of pregnancy. This is when the heart is developing from a simple tube-like structure into a shape more like a fully-formed heart.

#### **2. Genetic conditions**

Several genetic health conditions that a baby inherits from one or both parents can cause congenital heart disease. It's also recognised that certain types of congenital heart disease run in families.

Down's syndrome is the most widely-known genetic condition that can cause congenital heart disease.



Turner Syndrome and Noonan syndrome are other such genetic disorder.

Purification techniques should be given to both the parents before conception.

### 3. Maternal diabetes

Women with diabetes are five times more likely to give birth to a baby with congenital heart disease than women who don't have diabetes. This increased risk only applies to type 1 diabetes and type 2 diabetes. It doesn't apply to gestational diabetes, which can develop during pregnancy and usually disappears once the baby is born.

The increased risk is thought to be caused by high levels of the hormone insulin in the blood, which may interfere with the normal development of a foetus.

Diet regime has been explained for a pregnant lady so that such diseases can be prevented. Charak has mentioned that during pregnancy if mother indulges in excessive sweet intake it leads to prameha.

### 4. Alcohol

If a pregnant woman drinks too much alcohol during pregnancy, it can have a poisonous effect on the tissue of the foetus. This is known as foetal alcohol syndrome.

It's estimated that as many as half of all children with foetal alcohol syndrome will have congenital heart disease – most commonly, ventricular or atrial septal defects.

Similarly Smoking during pregnancy also has been linked to several congenital heart defects, including septal defects.

Contraindicated food items and lifestyle have been mentioned in ayurvedic classics which need to be followed.

## 5. Rubella

Rubella (German measles) is an infectious condition caused by a virus. It isn't usually a serious infection for adults or children, but it can have a devastating effect on an unborn baby if a mother develops a rubella infection during the first 8 to 10 weeks of pregnancy.

A rubella infection can cause multiple birth defects, including congenital heart disease.

All women of childbearing age should be vaccinated against rubella. Paripathadi kadha and such other medicines have been found to be useful clinically in such patients.

## 6. Flu

Women who get flu during the first trimester (three months) of pregnancy are twice as likely to give birth to a baby with congenital heart disease than the general population. The reasons for this are unclear.

The flu vaccine is recommended for all pregnant women.

## 7. Medications

There are several medications linked to an increased risk of a baby being born with congenital heart disease. These include:

certain anti-seizure medications – such as benzodiazepines and lithium

certain acne medications – such as isotretinoin and topical retinoids (see treating acne for more information)

ibuprofen – women who take the painkiller ibuprofen during the first trimester of their pregnancy are twice as likely to give birth to a baby with congenital heart disease than the general population

Paracetamol is a safer alternative, although ideally you should avoid taking any medicines while you're pregnant, particularly during the first three months of pregnancy.

#### 8. Phenylketonuria (PKU)

Phenylketonuria (PKU) is a rare genetic condition present from birth. In PKU, the body can't break down a chemical called phenylalanine, which builds up in the blood and brain. This can cause learning and behavioural difficulties.

PKU can usually be effectively treated with a low-protein diet and dietary supplements. Pregnant mothers with PKU who don't do this are six times more likely to give birth to a baby with congenital heart disease than the general population.

#### 9. Organic solvents

Women who are exposed to organic solvents are three times more likely to give birth to a baby with congenital heart disease than the general population.

Organic solvents are chemicals found in a wide range of products and substances, such as paint, nail polish and glue.

10. Gut bacteria has its impact on development of cardiac diseases especially contents such as lecithin and carnitine when not digested by gut bacteria leads to vascular diseases.

Food containing lecithin and carnitine are given below

Lecithin	and	Carnitine
Eggs		Poultry
Milk		Pork
Cream		Duck

Dairy

Lamb

Liver

Venison

Red Meat

Shell Fish Fish

Gut flora also known as gut microbiome has a unique role in digestion and absorption. Once the gut microbiome gets vitiated it leads to indigestion and malabsorption. The undigested food becomes substratum for bacterial overgrowth, the ecosystem of gut microbiome is affected and gut microbiome now starts producing toxins.

Further impaired digestion disturbs the peristalsis and in return retention of mala due to impaired evacuation of stools. The impaired evacuation of stools hampers the metabolism. The impaired metabolism leads to impaired gut microbiome and the latest research has shown that impaired gut microbiota can lead to various disorders, from heart disorders to psychological disorders. Thus a vicious cycle continues and it is clinically observed that patients give history of impaired digestion prior to heart disease. Thus a important preventive cardiology outlook can be given by maintaining better digestion and healthy gut bacteria

#### 11. Folic Acid and Homocystiene relationship

It is also observed that due to impaired digestion and absorption and diet having low nutritional values leads to nutritional deficiency especially folic acid which lead to increased homocystine levels, another cause for various serious diseases...

#### Acharya charak, acharya chakrapani and my approach to

**Vātaj Hridroga:** Vata is necessary for the coordinated functioning of heart. If vata gets vitiated the conduction defects may occur. Tachycardia and bradycardia are the 2 main classification of impaired cardiac conduction. Dara or dardarika as explained by Chakrapani or hridrava as explained by Yogindranath Sen explained the tachycardia. Various rhythmic and arrhythmic tachycardia have been



described by Modern science for eg. Sinus tachycardia, which originates from the sino-atrial (SA) node, near the base of the superior vena cava, Atrial fibrillation, Atrial flutter, AV nodal reentrant tachycardia, Accessory pathway mediated tachycardia, Atrial tachycardia, Multifocal atrial tachycardia, Junctional tachycardia, Ventricular tachycardia, any tachycardia that originates in the ventricles, Any narrow complex tachycardia combined with a problem with the conduction system of the heart, often termed "supraventricular tachycardia with aberrancy", A narrow complex tachycardia with an accessory conduction pathway, often termed "supraventricular tachycardia with pre-excitation" (e.g. Wolff—Parkinson—White syndrome).

Hridstambha explains the bradycardia. Stoppage, obstruction, suppression are various meaning of stambha. Sinus bradycardia, Sick sinus syndrome, AV block etc explain the condition of slow heart rate. Asystole, also known as flatline, is a state of no electrical activity from the heart and therefore no blood flow. It results in cardiac arrest. It may also be noted that tachycardia may later convert into asystole. Thus various condition resulting into tachy and bradycardia may be considered in vataj hridroga.

Near-fainting or fainting (murcha), Dizziness, Confusion or memory problems (pramoha/ sam moha) are the symptoms other than chest pain, Fatigue, Shortness of breath.

Hritshunyata explains the emptiness due to non filling of the heart chambers as desolateness is also meaning of sunyata or it may be understood as akinesia wherein no movement (asystole) is observed.

Ayurveda perspective; Hridaya gata vāta - Shālaparni shatāvari Arjuna brahmi kamal each 1 gm bid with milk or as ksheerapaaka. Prabhākar vati 125 mg bid , hridabasti should be done.. shirodhārā with brāhmi kvātha ; in tachyarrhythmia, thromboembolism is common complication, so , use of kamala ( better to use kamala kshāra) is helpful in preventing the thrombus formation.. Shālaparni

shatāvāri Arjuna help in controlling arrhythmia.. To maintain अव्याहत गति is prime concern..

On world Heart Day ; Vaivarnya , one of clinical features in hridroga mentioned by Acharya Charak in Ch. Chi. 26 , explains discolouration which the patient may present. Cyanosis is general presentation in cardiac diseases but other cutaneous presentation like lupus pernio, pink discoloration on the tip of nose, face etc which is generally a feature of sarcoidosis with cardio-pulmonary involvement. Livedo reticularis, is characterized by mottled, erythematous discoloration of the skin, which blanches on pressure. Livedo reticularis is the most common dermatologic manifestation of a cholesterol embolism. Erythema marginatum occurs in 10% of children with their first attack of acute rheumatic fever (ARF). Erythema marginatum is a flat to mildly elevated, pinkish, nonpruritic, transient eruption found primarily on the trunk and proximal extremities. Overall, it occurs in less than 5% of patients with rheumatic fever. Subcutaneous nodules are also rare in rheumatic fever but are associated with more severe carditis, as they usually present many weeks after the onset of disease; they are generally found over bony prominences and are usually painless because it can involve the pericardium, epicardium, myocardium, and endocardium. Xanthelasma palpebrarum in a patient with hyperlipidemia. Xanthelasma is Yellow flat plaques over the upper or lower eyelids, most often near the inner part of the eye. Hyperlipidemia exists in approximately 50% of patients with xanthelasma palpebrarum. Eruptive xanthomas are characterized by crops of 1- to 5-mm yellow-orange papules with surrounding erythema, most commonly on the extensor surfaces of extremities and the buttocks. This condition is most strongly associated with hypertriglyceridemia

Conjunctival pallor; Pallor in a patient with a prosthetic valve may be indicative of hemolytic anemia. Icterus is present in Right Heart Failure ,( presence of raised

JVP , Congestive Hepatomegaly, Hepatojugular reflux, and edema confirm the diagnosis of RHF ..).

Janeway lesions are associated with acute endocarditis, of which S aureus is the most common cause. Osler's nodes are associated with subacute bacterial endocarditis and S viridans. Osler's nodes are painful, erythematous nodules most commonly found on the pulp of fingers and toes.

Other cutaneous manifestations of infective endocarditis include splinter/subungual hemorrhages.

Vikriti varna urdhva sharir..ch.ind.. cyanosis in fingers... in Raynaud's disease.... Nayan neelvarna.ch.ind.. cyanosis retinae.. blue sclerae..in Emphysema.CHD with R--L shunt , osteogenesis imperfecta( bony dysplasia, teeth mal occlusion and bluish sclerae).....

Vikriti varna adhah sharirah.. ch.ind. that is cyanosis in toes not in fingers, also known as differential cyanosis.. e.g. in PDA (patent ductus arteriosus ) with reverse right to left shunt..

**Pittaj hridroga** includes infective conditions of cardiac diseases like infective endocarditis/ myocarditis/ pericarditis, Rheumatic heart disease, infective cardiomyopathy. Alcohol, chemotherapeutic drugs, heavy metals like arsenic etc induced heart disease has similarity with pittaj hridroga.

A small number of patients present with fulminant myocarditis, with rapid progression from a severe febrile (jwar) respiratory syndrome to cardiogenic shock that may involve multiple organ systems, leading to renal failure, hepatic failure (cause for pittata), and coagulopathy. The cardiogenic shock leads to cardiac syncope (tamo darshan/ moha). The myalgia due to infection leads to distress (satras). Breakdown of haemoglobin may also be the cause for yellow discoloration alongwith hepatic involvement. In tuberculous pericarditis, fever,



night sweats (sweda), and weight loss, were commonly noted. Acute pericarditis present as pain as if burning sensation (daha).

Hetus like lavana excess explains the rakta vikriti & vridhi which leads to hypervolaemia contributing to hypertension and thereby hypertensive cardiac disease.

**Kaphaja hridroga:** stabdha, supta and stimita are the 3 lakshana mentioned by Caraka. The Sanskrit meaning of the word stabdha is firmly fixed, stiff , rigid , immovable , paralyzed , senseless , dull, solidified, tardy , slack , slow whereas supta means insensible, dull, resting, latent, inactive and stimita means Fixed, rigid, unmoved, motionless, steady; paralysed, flowing gently along.

It means heart is motionless, inactive, slow, solidified, rigid. This 3 words extent the scope of kaphaja hridroga from non infective cardiomyopathy to cardiac tamponade.

Pulseless electrical activity (PEA), also known by as electromechanical dissociation, refers to cardiac arrest in which a heart rhythm is observed on the electrocardiogram that should be producing a pulse, but is not explain the supta , chakrapani says that supta is niskriyata. The stretching of myocardium is reduced resembles the stabdhata. Due to collection of fluid the steady motionless condition wherein the apex beat is not palpable or peripheral pulse is not palpable explains stimitata

Pulseless electrical activity leads to a loss of cardiac output, and the blood supply to the brain is interrupted. As a result, PEA is usually noticed when a person loses consciousness (suptata) and stops breathing spontaneously. The cool touch resembles stimitata and the heaviness felt is ashmavrita. Ashmaavritam as pericardial calcification is observed in constrictive pericarditis on echocardiographic study..



6 Nov 2014

Some specific ECG findings in ventricular tachycardia ( VT); Brugada's sign- the distance from onset of QRS complex to nadir of S-wave is >100 ms. Josephson's sign ; notching near nadir of S-wave.... RSR' complexes with taller "left rabbit ear" this is most specific findings in favour of VT.. This is in contrast to RBBB where "right rabbit ear" is taller...Very broad complexes >160 ms.. regular rhythm, originates from single focus within ventricle, produces uniform QRS complexes within each lead in monomorphic VT..

**Terminalia arjuna** \* \* few scientific knowledge .

Significant reduction in anginal frequency.

significant improvement in left ventricular ejection fraction.

reduction in left ventricular mass on echocardiography following three months of therapy.

patients with ischaemic cardiomyopathy showed significant symptomatic relief in coronary heart failure from NYHA class III to NYHA class I.

increase in the coronary flow. The inotropic effect is considered to be mediated through the high concentration of  $Ca^{++}$  present in the plant.

Aqueous and alcoholic bark extract, when given intravenously, intracerebrally, and intravertebrally in dog, resulted in a dose-dependent decrease in blood pressure.

Dried, pulverized bark has been shown to augment endogenous antioxidant compounds of rat heart and prevent oxidative stress associated with ischemic-reperfusion injury of the heart.

Arjuna bark extract has a significant prophylactic and therapeutic beneficial effect in protecting heart against catecholamine-induced CHF, possibly through maintaining endogenous antioxidant enzyme activities and inhibiting LPO and cytokine levels.

triterpenoids derived from arjuna extract containing arjunolic acid show cardioprotective activity by boosting endogenous antioxidant defense system.

The hypolipidemic action is thought to be mediated through increased hepatic clearance of cholesterol, down-regulation of lipogenic enzymes, and inhibition of HMG-CoA reductase.

significant improvement in diastolic dysfunction..

Arjuna also caused significant inhibition of platelet aggregation.

A significant reduction in lipoprotein(a) levels amounting to 24.71% following the administration of arjuna .

hydroalcoholic extract of bark when given for 2 weeks led to significant regression of the endothelial abnormality amongst smokers.

the methanol extract was found to possess significant thrombolytic activity (30.57%). It also significantly inhibited the hemolysis of RBCs in both hypotonic solution and heat-induced conditions.

### **Treatment (Samprapti vighatana) approach to Hypertension**

Why to use tikta rasaatmaka and kashaaya rasaatmaka dravya in combination ?

Excess salt intake is one of cause of Hypertension , the mechanism is through activated RAAS . Ayurved perspective : lavanam pittam prakopayati raktam vardhayati cha (one aspect ) : hypervolemia - increased venous return - increased stroke volume - increased cardiac output - Hypertension.. kashaaya rasa is antagonist to lavana , but it's alone not so effective in reducing blood pressure , so , after adding certain tikta dravya , reduction in both systolic & diastolic pressure is observed .. kashaaya rasa is enriched in vaayu and prithvi , tikta rasa is dominated by vaayu , aakaasha ,and lavana rasa is dominated by aap and agni .. vaayu and prithvi of kashaaya rasa help in decreasing fluid volume , but can't work on total peripheral resistance ( an other aspect ) , therefore , tikta rasa is added , and aakaasha mahaabhoota with its apratighaatatva Guna decrease the pratighaatatva ( total peripheral resistance ) . Lavanam kapham prakopayati and in turn leads to vasoconstriction with or without dhamanipratichaya . Conclusion : it's tridoshaja vyaadhi with rasaraktadi ambu dhātu dushti.. my choice of drugs : Arjun shatavari brahmi jatamansi shankhapushpi punarnava gokshuru varuna vacha and elā.. I use this combination with or without sarpagandha..

Hypertension is modern time diagnosed disease with multiple causes and effects on multiple organ-systems.. Hriday, raktavaha srotas and rakta dhaatu are main organ/system interplay to induce HTN. Hridaya is derived from; Hri means aaharana/venous return / preload, Da means dadaati / stroke volume /afterload and ya means ate /gati/contractility of myocardium +heart rate These three are basic components to determine the function of hridaya.. aaharana/aadaana karma is due to praana vaayu. Dadati /stroke volume is due to vyaana vaayu.. ate /gati is due to udaana vaayu because of its prayatna karma /action potential of cardiac muscles.. Heart rate and rhythmicity is due to praana udaana and vyaana..

Vasodilation and vasoconstriction are related to perfusion / rasa vikshepana , so its due to vyaana vaata..The role of renin-angiotensin -aldosterone system (RAAS ) is to regulate salt and fluid volume and to maintain blood pressure on long term , its related with apaana vaayu as this system excretes sodium and in turn water..Rasa vikshepana is karma of vyaana , as per acharya chakrapani (on ch.chi 15/36) rasa means rasaraktaadi ambu dhatu.. so vikshepana of rasaraktadi ambu dhatu is due to vyaana vaayu..

The role of vasocenter is to regulate blood pressure so its praana vaayu karma..

Fluidity/dravtva of rakta dhaatu is also responsible for aaharan , and vikshepana . So there is importance of rakta dhaatu/rasaraktaadi ambu dhaatu.. vikshepana occurs through srotas so there is role of raktavaha srotas.. acharya charak mentions " Anupahata sarva dhaatooshma maruta srotah (ch.su.28/3) . Acharya chakrapani ; maruto vaa dhatuposhaka rasavaahi vyaana rupah ; shows the role of vyaana vaata is also crucial in dhaatu nirmiti and dhaatu parivahana or vikshepana.. These are basic components participate in pathogenesis of HTN..As per understanding of metabolic syndrome , the role of kapha and saama meda are clear in pathogenesis of DM , IHD , Obesity ,Dyslipidaemia and HTN. The role of pitta , One example ; Salt intake - pitta prakopa & rakta dhaatu vridhi ( as acharya charak mentions lavano rasah pittam kopayati and raktam vardhayati (ch.su.26/42/\*3) hypervolemia - increased cardiac output ; HTN ( BP  $\leftrightarrow$  CO  $\times$  TPR )..

The role of kapha; Dhamanipraticay - increased total peripheral- resistance (TPR) - HTN..

Few example for vaata bheda.. : Kaama shoka bhayat vaayuh - praana vaayu prakopa - stimulation of central sympathetic outflow/vasomotor activity - increased heart rate in turn increased cardiac output and increased TPR - HTN. Aortic stenosis ; increased afterload - increased vyaana vaayu karma -HTN..



Tachycardia - increased contractility of myocardium - increased prayatna karma of udaana vaata - HTN..The role of higher center ; use of medhya drugs , The role of hridaya : use of hridya drugs , The role of dhamanipratichaya - kapha and saama meda - tikta and katu rasa dravya . The role of RAAS - use of mootral drugs , anulomana drugs.. the role of samaan - use of deepaniya ghrita(?)/drugs, The role of rakta - use of mootral drugs / pitta shaman/virechan.. etc..

Glomerulonephritis - increased RAAS - salt and water retention , Vasoconstriction and increased heart rate - HTN ( Apaana vaayu vaishamya) Insulin resistance - hyperinsulinaemia - vascular smooth muscles hypertrophy and increased entry of ca ions in endothelium of vascular smooth muscles - contraction of SM -vasoconstriction - HTN \*( Role of samaana and vyaana ) . Rasa means rasa raktaadi ambu dhatu - moola of rasavaha srotas is hridaya so ashraya ashrayi bhava shows involvement of rasa dhatu. Atishthaulya - medodhatu pradoshaj vikar - meda sahitam aamam vaataadinaam rodham gyeyam ( dhamanipratichaya).

Free fatty acid flux to liver is associated with increased production of apo B-containing, triglyceride rich VLDLs. Hypertriglyceridemia is an excellent marker of insulin resistant condition.. so t/t is medoghna drugs or as per prameha. In dhamanipratichaya , initial dosh is kapha but later vyaana vaata becomes impaired to induce HTN.. t/t - Kaphaghna /medoghna

First, nervous stimulation - hormones - receptors - influx of ions -action...

Beta or alpha receptors mediate the action of intermediate metabolite/energy/influx of ions , so vaata predominance.

Osmoreceptors , atrial natriuretic peptide etc also interplay.. therefore, as i think , all controllers/regulators/modulators are vaata dosha related..: There is low renin , normal renin , high renin condition in HTN.. Calcium supplements help in reducing HTN and calcium channel blockers also reduces BP...Therefore i think the

role of multiple factors in HTN , including vaata , pitta , kapha, rakta medaadi... and manas too....( as yoga is found effective )...

### **The series of understanding of Hypertension :**

(1)

Panchātmā vāta : the understanding helps to go through Hypertension with ayurveda perspective ;

The functional status of Vāta with its sub units can be better understood by analyzing certain physiological events. The normal electrical conduction in the heart allows the impulse that is generated by the sinoatrial node (SA node) of the heart to be propagated to, and stimulate, the cardiac muscle (myocardium). The myocardium contracts after stimulation. It is the ordered, rhythmic stimulation of the myocardium during the cardiac cycle that allows efficient contraction of the heart, thereby allowing blood to be pumped throughout the body. Signals arising in the SA node (located in the right atrium) stimulate the atria to contract and travel to the AV node, which is located in the interatrial septum. After a delay, the stimulus diverges and is conducted through the left and right Bundle of His to the respective Purkinje fibers for each side of the heart, as well as to the endocardium at the apex of the heart, then finally to the ventricular epicardium.

On the microscopic level, the wave of depolarization propagates to adjacent cells via gap junctions located on the intercalated disk. The heart is a functional syncytium (not to be confused with a true "syncytium" in which all the cells are fused together, sharing the same plasma membrane as in skeletal muscle). In a functional syncytium, electrical impulses propagate freely between cells in every direction, so that the myocardium functions as a single contractile unit. This is the avyāhata gati of vāta which is necessary for the rapid, synchronous depolarization

of the myocardium. Conduction from SA to AV to bundles and Purkinje fiber is the *aparityakta swa mārṅa* of *vāta*. This rhythmical and conductive system of the heart is susceptible to damage by heart disease, especially by ischemia of the heart tissues resulting from poor coronary blood flow. The result is often a bizarre heart rhythm or abnormal sequence of contraction of the heart chambers, and the pumping effectiveness of the heart often is affected severely, even to the extent of causing death. This explains the *vyāhata gati* of *vāta* which is the cause of death.

The circulatory system is the main method for blood transportation within body. This system is a complex highway of vessels, and its main purpose is to move blood and nutrients throughout body. The circulatory system is also responsible for exchanging gases and removing waste products from body. Unlike an open circulatory system, a closed circulatory system is more structured and controlled. The blood of a closed system always flows inside vessels. These vessels make up the plumbing circuit of the body and can be found throughout the entire body. This plumbing circuit can be broken down into three different types of vessels, or tubes that transport blood throughout the body: arteries, capillaries and veins. Thus a continuous flow of blood from Left ventricles to the aorta to arteries all over the body then to arterioles into capillaries into venules into veins and back to the right atrium then right ventricle via pulmonary artery to the lungs and via pulmonary veins to the left atrium and back to left ventricle. This is how blood is propagated from heart to the periphery and back to the heart. The modern explanation resembles Caraka explanation as mentioned in *Ca. Ci. 15/36*

This function of *vāta* is *swa sthānastha* which helps to maintain the homeostasis or *swāsthya* but when *avarodh* to this *gati* takes place may be due to any reason the *swa mārṅāsthita vāta* gets *vimārṅa gata* as explained in *samprapti* of *śōtha* (*Ca. Ci. 12/8*).

Various edemas are either due to excessive secretion (*apāna vāyu*) or reduced absorption (*prāna vāyu*) as understood in *samprapti* of *udara*. Disturbed

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concentration of solutes and solvents causes changes in pressure (vyāna vāyu) either intravascular or extra vascular. The electrolyte balance is brought about by sweda dōṣa ambu srotas sthāyi vāyu i.e. samāna vāyu.

Prakruti sthita vāta is the one which is akshina vridha:

Reduced respiratory rate due to depressed respiratory centre explains kshina prāna vāyu whereas vridha prāna vāyu may be one of the causes for increased ventilation.

Prayatna, urjā are functions of udāna vāyu. Excessive excitation of cell due to excess action potential explains the vridha udāna vāyu whereas inhibition of cell activity due to reduced action potential is due to kshina udāna vāyu.

Excessive stimulation of agni (atyagni) causes increased appetite one reason being vridha samāna vāta whereas agnimāndya, grahani etc may be caused by decrease stimulation of agni by samāna vāta.

Normal pulse rate ranges from 60-80/min. Excessive pulse rate explains the repeated contraction of heart one of the cause being excessive ākunchan prasārana karma of vyāna vridhi whereas one of the cause of bradycardia may be kshina vyāna vāyu.

Increased peristalsis is the cause for increased frequency of stools one of the reason being vridha apāna vāta whereas reduced peristalsis causes constipation one reason being kshina apāna vāta...

Continued ....

Cell modulation : the role of panch vāta ;

(2)

The selective permeability of the plasma membrane allows a living cell to maintain different concentrations of certain substances on either side of the



plasma membrane. A concentration gradient is a difference in the concentration of a chemical from one place to another, such as from the inside to the outside of the plasma membrane. Many ions and molecules are more concentrated in either the cytosol or the extracellular fluid. For instance, oxygen molecules and sodium ions (Na) are more concentrated in the extracellular fluid than in the cytosol; the opposite is true of carbon dioxide molecules and potassium ions (K). The plasma membrane also creates a difference in the distribution of positively and negatively charged ions between the two sides of the plasma membrane. Typically, the inner surface of the plasma membrane is more negatively charged and the outer surface is more positively charged. A difference in electrical charges between two regions constitutes an electrical gradient. Because it occurs across the plasma membrane, this charge difference is termed the membrane potential. In many cases a substance will move across a plasma membrane down its concentration gradient. That is to say, a substance will move "downhill," from where it is more concentrated to where it is less concentrated, to reach equilibrium. Similarly, a positively charged substance will tend to move toward a negatively charged area, and a negatively charged substance will tend to move toward a positively charged area. The combined influence of the concentration gradient and the electrical gradient on movement of a particular ion is referred to as its electrochemical gradient.

Transport of materials across the plasma membrane is essential to the life of a cell. (āyū is one of the paryāya of vāyū). Certain substances must move into the cell to support metabolic reactions (pravesakrita karma of prāna vāyū). Other substances that have been produced by the cell for export or as cellular waste product (niskramana karma of apāna vāyū) must move out of the cell.

The concentration gradient which is maintained is essential for cellular activity. Resting membrane potential and active membrane potential are maintained at specific levels. For e.g. Charges of -90 mv is the resting charge which reaches to +35 mv when depolarized in cardiac cell thus this knowledge of potential gradient

is due to budhi dharan karma of prāna which cause the pumping of Na/K pump to activate. Thus knowledge of concentration gradient is karma of prāna vāyu. Further prāna means prinana ādāna karma i.e. helping entry/ facilitation of such ions, essential requirements within cell which will do prinan /poshan is also due to prāna. Thus process that initiates endocytosis is prāna vāyu.

Substances generally move across cellular membranes via transport processes that can be classified as passive or active, depending on whether they require cellular energy. In passive processes, a substance moves down its concentration or electrical gradient to cross the membrane using only its own kinetic energy. The continuous movement resembles the cala guna, a common quality of all the types of vāta. Modern describes it as the Brownian movement of the ions. Kinetic energy is intrinsic to the particles that are moving. There is no input of energy from the cell. An example is simple diffusion.

In active processes, cellular energy is used to drive the substance “uphill” against its concentration or electrical gradient. The cellular energy used is usually in the form of ATP. It explains the prayatna karma of udāna vāyu which is responsible for the activity. An example is active transport. Active transport is considered an active process because energy is required for carrier proteins to move solutes across the membrane against a concentration gradient. Two sources of cellular energy can be used to drive active transport: (1) Energy obtained from hydrolysis of adenosine triphosphate (ATP) is the source in primary active transport; (2) energy stored in an ionic concentration gradient is the source in secondary active transport. Like carrier-mediated facilitated diffusion, active transport processes exhibit a transport.

Many of the infolding of the inner membrane form shelves on which oxidative enzymes are attached. In addition, the inner cavity of the mitochondrion is filled with a matrix that contains large quantities of dissolved enzymes that are necessary for extracting energy from nutrients. These enzymes operate in association with the oxidative enzymes on the shelves to cause oxidation of the

nutrients, thereby forming carbon dioxide and water and at the same time releasing energy. The liberated energy is used to synthesize a “high-energy” substance called adenosine triphosphate (ATP). ATP is then transported out of the mitochondrion, and it diffuses throughout the cell to release its own energy wherever it is needed for performing cellular functions. Thus the phenomenon which triggers the oxidative process is the samāna vāyu which stimulates the oxidation i.e. role of agni.

The intracellular movement of proteins, ATP transfer, and vesicle transportation can be understood as the vyāpan/ vyuhan karma of vyāna vāyu.

The end metabolites formed within the cell are removed through the process of exocytosis. The process is initiated by apāna vāyu which helps in excretion, mokshan, munchan karma at the level of cell.

3

(4)

Amlam lavanam katuko cha pittam prakopayati ; its initial effect of these rasa . Amlam raktam dooshayati , lavanam raktam vardhayati , katuko shonita sanghaata bhinatti.. next step of these rasa .. now , specifically , lavanam pittam kapham cha prakopayati & raktam vardhayati (\* not dooshayati \*- refer to ayurved sellers for going through Charak samhita , not only ashtanga ).. flow , pressure , and resistance are 3 important factors of blood circulation.. flow of blood is either due to rasati iti raso drava dhaatu , tena roodhiraadinaamaapi dravaanaam grahanam bhavati ( blood volume ).. or vyaanen ( stretching of myocardium ; preload ) , these two factors help in venous return of blood.. first is blood volume.. lavanam raktam vardhayati - hypervolemia - increased venous return - increased stroke volume - increased cardiac output - increased blood pressure , but before it , feedback mechanisms work through osmoreceptors , thirst center ,atrial natriuretic peptide , brain natriuretic peptide , renin - angiotensin - aldosterone system to maintain osmolality of blood , but due to



result of non modulation ( vikaara vighaata abhaava ) Hypertension develop .. salt restriction is first option .. Diet approach to stop Hypertension (DASH) strictly prohibited extra salt consumption.. it's sodium chloride , not sodium alone , cause Hypertension.. mootrala dravya is still first choice in mild Hypertension , because of its natriuretic effect.. there must be therapeutic response data collection whether punarnava or gokshuru or both in combination are good , especially considering potassium levels in blood.. in Hypertensive patients , serum electrolytes should be checked before and later at regular intervals , especially serum potassium levels.. an other benefit of this blood test is to rule out hyperaldosteronism : Hypertension with hypokalaemia..

Will be continued...

(5)

Pressure is generated by heart / hridaya.. so , now , to discuss about hriday ;

Nirukti ; Haraterete hrida shabdah .. Shatapata bramhana...

HRI means āharana , to receive , venous return , preload.

DA means Dānārthaka , to give , stroke volume , afterload..

YA means Gatyārthaka , Rhythmic myocardial contraction , strength of myocardial contractility & heart rate..

Preload , afterload , contractility of myocardium & heart rate are major determining factors for cardiac output . For understanding of physiology of cardiac output , these 4 factors are to be explained in ayurved perspective , then , the role of pressure in circulation will be well elaborated..

To be continued.....!!

(6)

The role of Apāna vāta :

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The function of apāna vāta is dhārana ( su.su.15/4) . The apāna vāta acts to eliminate the Kitts - when it's excessive or natural urge for Kitts - elimination is present and keeps to retain when they are deficient or no urge is present . Acharya chakrapani on ch.si. 9 : although the place of apāna is medhrādi , but it may be hridaya , because -

Hridayāvyatiriktānuvidhāyitvāt hridayāshrita uchyate.

Hridaya plays a role in circulation of rasaraktadi ambu dhātu through vyāna vāta , but as apāna is responsible to eliminate the Kitta , it's apāna which transports the Kitta to be excreted through orifices..

In vāgbhatta , hridagada is mentioned due to apāna Vega vidhārana : indicates that the decreased elimination of Kitta from body can influence the hridayastha karma and subsequently may develop hridroga.

Modern science also accept the role of inadequate removal of intermediate metabolites ( pyruvate , lactate , etc ) consequent to hypoperfusion of myocardium leading to increased acidic pH of myocardial cells and in turn contribute to ischemia and later necrosis of myocardial cells..

Kapalan has hypothesized that volume expansion due to high sodium intake will lead to hypercalciurea , secondary hyperparathyroidism , and subsequent Hypertension. Increased dietary calcium intake would then restore parathyroid hormone levels to Normal and thus reduce blood pressure. This explanation shows the role of Apāna vāta in Hypertension.

Apāna vāta is regulating mechanism participate in elimination of Kitta or undue substances or excreta from the body through orifices. It's apāna vāta with crucial role in maintaining homeostasis of electrolytes and water balance in body.. non modulation or vikāra vighāta abhāva through Activated RAAS and in turn the development of Hypertension , edema , etc..

Udānam yojayet urdhvam apānam cha anulomayet. Ch.chi.28/218

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Apānenāvrite sarvam deepanam grāhi bheshajam ,

Vātānulomanam yachcha pakvāshayavishodhanam. Ch.chi.28/242-243..

Understanding of pathophysiology in specific condition will help in perfect treatment plan..

Thanks again for healthy readership .. ???????

Will be continued....

(7)

Hridaya is aerobic organ , which is exclusively dependent on energy derived from nutrients. Hridaya is place of ātmā , mana , oja , indriya , prāna vāta , vyāna vāta , sādha pitta , avalambaka kapha , etc , and as rasavaha & prānavaha srotomoola.. as per āshraya - āshrayi bhāva , all these factors and hridaya may influence each other.. for it's rhythmic contractions and relaxations , it requires the delicate balance between supply and demand or energy and workload.. Hridaya is supplied by dhamani , by which it gets nutrients and oxygen for its function. Vyāna is responsible for dhātuposhaka rasa vikshepana for hridayastha dhātukarma. The transportation of rasaraktādi ambu dhātu is also dependent on dravatva , and patency of srotas or dhamani , the autoregulation is very intact to maintain supply as per demand in healthy individuals. The regulatory mechanisms are comprised of central nervous system , autonomic nervous system , and local autoregulation.. Brain - heart , Heart - brain relationship is behind maintaining homeostasis. Hridaya, embryologically , is made up of kapha and rakta. Kapha is enriched in prithvi & āpa mahābhūta and rakta is enriched in agni mahābhūta , so , these 3 mahābhūta are important factors in genesis of hridaya.. Action potential in cardiac cells is unique , because of atrial & ventricular syncytium : there is 2 separate pumping chambers.. on the principle of concentration gradient pārvānsha move across cell membranes , and by the prayatna karma of udāna , the ions move opposite to their concentration gradient , āpa helps in both ways of

transportation. The action potential provide energy ( urjā) and eventually helps in strengthening the contractility of myocardium ( Bala ) , and with the help of vyāna vāta , contraction and relaxation occurs spontaneously , rhythmically under control of prāna vāta. Samāna vāta works as second messengers to activate oxidative enzymatic activities , and apāna helps in elimination of Kitta from Myocardial cells.. the structural or functional impairment of hridaya : kurvanti hridaye bādhām , hridirogam Tam prachakshate ..

We need such research in ayurved : below reference is posted as answer of a question asked by my one Student that which NSAIDs are safe in hypertensive patients with osteoarthritis?..

Scand J Rheumatol Suppl. 1986;62:36-40.

The arthritic patient with hypertension: selection of an NSAID.

Spence JD.

#### Abstract

Vasodilator prostaglandins produced in the renal medulla have a role in blood pressure regulation, beyond modulation of sodium and water retention. Systemic vasodilation resulting from effects of renomedullary prostaglandins lowers systemic vascular resistance, and administration of NSAIDs elevates blood pressure in hypertensive patients treated with diuretics and/or beta blockers, in patients with myocardial infarction, and in patients taking sympathomimetic agents such as phenylpropanolamine. Aspirin, which appears in the urine as salicylic acid (which has no effect on cyclooxygenase) has not been implicated as a drug which attenuates blood pressure control. Similarly, sulindac, the active sulfide metabolite of which is not filtered, does not inhibit renal synthesis of prostaglandins, though given in doses sufficient to inhibit serum thromboxane and 6-keto PGF 1-alpha. In a double-blind complete crossover study of blood

pressure and renal function in hypertensive patients controlled with timolol-hydrochlorothiazide, sulindac lowered blood pressure significantly, whereas naproxen and piroxicam significantly raised blood pressure, in the absence of any effect on GFR, plasma renin, weight, creatinine clearance, or urinary sodium. It is suggested that for arthritic patients with hypertension, the NSAIDs of choice are aspirin and sulindac.

PMID: 3541167

To be continued..

Acharya charak , acharya chakrapani , and modern time interpretation;

Sharira kledam punardooshayan mootratvena parinamayati , mootravahaanaam cha srotasaam vankshanabastiprabhavaanaam medahkledopahitaani guruni mukhaanyaasaadya pratiroodhyate . Ch.ni . 4/8 , aasaadya pratiroodhyate iti gatvaa avatishthate :(acharya chakrapani ..

Osmotic diuresis is the increase of urination rate caused by the presence of certain substances in the small tubes of the kidneys. The excretion occurs when substances such as glucose enter the kidney tubules and cannot be reabsorbed (due to a pathological state or the normal nature of the substance). The substances cause an increase in the osmotic pressure within the tubule, causing retention of water within the lumen, and thus reduces the reabsorption of water, increasing urine output (i.e. diuresis). The same effect can be seen in therapeutics such as mannitol, which is used to increase urine output and decrease extracellular fluid volume.

Substances in the circulation can also increase the amount of circulating fluid by increasing the osmolarity of the blood. This has the effect of pulling water from the interstitial space, making more water available in the blood and causing the kidney to compensate by removing it as urine. In hypotension, often colloids are used intravenously to increase circulating volume in themselves, but as they exert



a certain amount of osmotic pressure, water is therefore also moved, further increasing circulating volume. As blood pressure increases, the kidney removes the excess fluid as urine. Sodium, chloride, potassium are excreted in Osmotic diuresis, originating from Diabetes Mellitus (DM). Osmotic diuresis results in dehydration from polyuria and the classic polydipsia (excessive thirst) associated with DM.}}

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(8)

An article in favour of my approach that rakta vridhhi (qualitative &/or quantitative ) is one of causes in Hypertension . Because of heterogeneous disease , I consider specific dosha - dooshya involvement in hypertensive individual. Sarva Eva bhavantah samyagāhuranyatraikāntikavachanāt : ācharya ātreya ( ch.soo. 12/13).

Arch Mal Coeur Vaiss. 1985 Oct;78(11):1706-9.

[Blood hyperviscosity syndrome in essential arterial hypertension. Characterization and clinical effects].

[Article in French]

Zannad F, Medeiros C, Voisin P, Bruntz JF, Stoltz F, Gilgenkrantz JM.

Abstract

Blood viscosity (BV) is one determinant of total arterial resistance (TAR) which is usually increased in hypertension. This increase is mainly related to vasoconstriction. In this study, we investigated the blood rheological properties in hypertension and their relation to blood pressure (BP) and left ventricular

hypertrophy (LVH) since the latter could be related to increased TAR. BP and echocardiographic measurement of left ventricular mass (LVM) according to Devereux were measured and blood samples obtained from 22 untreated hypertensives aged 31 to 62 (13 men, 9 women) Control group consisted of 30 age-matched, normotensive blood donors. Results are shown in table I. (Formula: see text). A positive significant correlation was found between LVM and BV ( $r = 0.50$ ;  $p$  less than 0.05) red cell filterability ( $r = 0.53$ ;  $p$  less than 0.05) and red cell aggregability ( $r = 0.57$ ;  $p$  less than 0.02). These results suggest that erythrocyte abnormalities are one of the determinants of the hyperviscosity syndrome in hypertension. Some variables of this syndrome were related to LVM and could therefore be among the determinants or a consequence of LVH in Hypertension.

Ayurved perspective in hyperviscosity syndrome ;

As understood in grahani adhyaya yugpat, sarvatra, continuous vikshepa of rasa rupa dhatu is by vyanvayu which is responsible for nutrition of sthyayi dhatu. Whenever due to kha vaigunya the vikshepita rasa gets obstructed/ stagnated, pathogenesis takes place. Thus for avyathagati of vata normalcy of marga and margastha dhatu is essential.

Marga means various channels, srotas, sira, dhamani, rasayani, rasavahini, nadi, pantha, sharirchidra, aashaya, niketa etc. whereas margastha dhatu means drava rupa asthayi raktadi dhatu.

Specific ratio of pancamahabhut maintains the dravatva of dhatu. More the viscosity slow is the flow of the drava rupa dhatu. Viscosity will increase whenever parthivata will increase in proportion in margastha dhatu. Change in specific proportion is primarily due to agni. It may be at level of jatharagni, dhatvagni or bhutagni. Secondly, anupahat agni is responsible for maintaining of normalcy of dhatus. Agnimandya leads to apachit dhatu vridhi. Such apachit dhatu are nothing but aam which may act as antigen. Vyadhi vighatkar bhava comes in action to prevent adherence of aam with specific dhatu.

Thus presence of aam and vyadhi vigatkar bhava changes the specific ratio of dravatva leading to reduction of flow (Saratva) or capillary perfusion and increase in organ congestion and syndromes of hyperviscosity.

(9) ;

Potassium and calcium in Hypertension ;

its known that potassium-rich foods can help control high blood pressure. A variety of foods contain potassium, such as fruits, vegetables, dairy products, and fish. Those especially rich in potassium include pomegranates , potatoes, lima beans, bananas, tomato sauce, beet greens, fat-free yogurt or milk, halibut, tuna, and orange juice.

One cup of orange juice: 496 milligrams (mg) of potassium

One baked potato: 1,081 mg

One cup of sliced bananas: 594 mg

One cup of tomato sauce: 909 mg

One cup of cooked spinach: 839 mg

Recommendations are to eat about 4,700 mg of potassium each day. However , too much potassium can be especially dangerous for older adults and people with kidney disorders.

The Scoop on Calcium and Hypertension:

A recent review of research on calcium to treat high blood pressure examined 13 small studies and found little evidence that calcium supplements helped to reduce hypertension, though the authors of the review did point out that the

studies may not have been big enough to draw good conclusions and more research is needed.

Even though there is no conclusive evidence that calcium supplements will help to control hypertension, at least one large study found that a low-fat diet that included dairy products (a rich source of calcium) did decrease the risk of developing hypertension for a study group of almost 30,000 women over the age of 45.

This research showed that women who drank two or more daily servings of skim milk (or consumed other low-fat dairy products) reduced their risk for developing high blood pressure by 10 percent compared with women who didn't consume dairy products as frequently. It wasn't clear if it was the calcium or the consumption of dairy products in general that tended to lower a person's risk for developing high blood pressure. (The study also found that taking calcium as a supplement didn't have the same benefit.)

Pomegranate juice ;

Pomegranate juice is in the running as the most heart-healthy juice. It appears to protect the heart and arteries.

Small studies have shown that the juice improves blood flow and keeps the arteries from becoming stiff and thick. It may also slow the growth of plaque and buildup of cholesterol in the arteries. But pomegranate may react negatively with blood pressure and cholesterol medications like statins.

Drinking pomegranate juice daily may also help lower systolic blood pressure. But more studies need to be done to determine if pomegranate juice can decrease overall blood pressure in the long term.

To be continued...



(10)

#### Milk and Hypertension ;

Nine milk protein substrates were hydrolysed in vitro with five proteases for various times (0, 3, 6, and 24 h), and the angiotensin-converting enzyme (ACE)-inhibitory activity of hydrolysates was assessed. Overall, the casein substrates gave rise to hydrolysates with significantly higher ACE-inhibitory activity than the whey protein (WP) substrates (85% vs. 79%). No significant difference between 3 and 24 h of hydrolysis was found. A reasonable correlation was found between the ACE inhibition of the 6 h hydrolysates determined in vitro and estimated by in silico modelling. The highest ACE-inhibitory activity was found in hydrolysates made with thermolysin followed by proteinase K, trypsin, pepsin and *Bacillus licheniformis* protease. The IC<sub>50</sub> values for thermolysin hydrolysates of caseins and WPs were 45–83 and 90–400 µg mL<sup>-1</sup>, respectively, with α-lactalbumin giving the highest inhibitory activity. Thermolysin, proteinase K and trypsin were useful for the release of highly potent ACE-inhibitory peptides from both WPs and caseins.

#### Magnesium and Cardiac Action Potential;

Magnesium (Mg<sup>++</sup>) is the second most abundant intracellular ion.

Normal Serum Mg<sup>++</sup> is 1.8 to 2.5 mg/dL or .8 to 1.5 mmol/L (millimoles per liter).

(This values may vary depending on sources)

Keep in mind, the Mg<sup>++</sup> concentration in the average adult is approximately 25g, but most of our Mg<sup>++</sup> is found in bones and intracellular. Because this makes it hard to assess the true Mg<sup>++</sup> concentration, true Mg<sup>++</sup> measurement is often not performed, instead, Serum Mg<sup>++</sup> levels are obtained. This measurement does not

fully correlate with overall  $Mg^{++}$  because only a small amount is found in the serum, usually approximately 1% of all  $Mg^{++}$ .

$Mg^{++}$  has over 300 different physiologic functions, and it affects multiple phases of the cardiac AP.

$Mg^{++}$  acts as a physiologic Calcium ( $Ca^{++}$ ) Channel inhibitor by slowing slow L-Type Calcium channel during PHASE 2 of the AP.

This reduces further  $Ca^{++}$  release by the Sarcoplasmic Reticulum which leads to reduced automaticity, contractility and conductivity through cardiac tissue, including the AVN.

Hypomagnesemia (Serum  $Mg^{++}$  < 1.8 mg/dL or .8 mmol/L).

$Mg^{++}$  mediates Potassium ( $K^{+}$ ) influx during PHASE 4 of the AP, therefore, during Hypomagnesemia,  $K^{+}$  influx is partially inhibited, which leads to delayed ventricular repolarization.

Because  $Mg^{++}$  also is responsible for proper  $Na^{+}/K^{+}$  pump, Hypomagnesemia leads to  $K^{+}$  loss which leads to Hypokalemia (serum  $K^{+}$  < 3 mEq/L).

Whang et al studied 46 Hypokalemic patients who also presented with Hypomagnesemia. In these cases, the Hypokalemia was only corrected when the associated Hypomagnesemia was fixed.

Common Hypomagnesemia causes include:

- Alcoholism
- Diabetic Ketoacidosis
- Malnutrition
- Digoxin
- Diuretics (e.g. Thiazides, Loop Diuretics)

ECG Changes consistent with Hypomagnesemia:-

ST segment depression (horizontal or downsloping ST segment).

Tachycardia leading to bradycardia

Diminished T wave amplitude or flattened T waves

Presence of U waves (associated with Hypokalemia)

Widened QRS complex >100ms (rare)

Prolonged QTc (due to repolarization delay)

Prolonged PR interval

Torsade De Pointes (Polymorphic Ventricular Tachycardia)

hypomag-bmp

- ST depression in V3-6 and Leads II and III

- Diminished T waves

- Serum Magnesium = 1.5 mg/dL

- Serum K<sup>+</sup> = 3.7 mEq/L

II& V2

- Flattened T waves

- Prolonged QT appearance due to prominent U wave

- Serum K<sup>+</sup> = < 2 mEq/L

torsades

- Torsade De Pointes

(11)

#### Magnesium and Hypertension :

the balance between Na: k , Ca: Mg ratio in extracellular and intracellular compartments help in maintaining blood pressure..

Magnesium status has a direct effect upon the relaxation capability of vascular smooth muscle cells and the regulation of the cellular placement of other cations important to blood pressure - cellular sodium:potassium (Na:K) ratio and intracellular calcium ( $iCa^{2+}$ ). As a result, nutritional magnesium has both direct and indirect impacts on the regulation of blood pressure and therefore on the occurrence of hypertension. Hypertension occurs when cellular Na:K ratios become too high, a consequence of a high sodium, low potassium diet or, indirectly, through a magnesium deficient state which causes a pseudo potassium deficit. Like wise, magnesium deficiency alters calcium metabolism, creating high  $iCa^{2+}$ , low serum calcium and low urinary calcium states even when calcium intake is adequate. High  $iCa^{2+}$  and high cellular Na:K ratio both occur when cellular magnesium becomes too low and the Mg-ATP driven sodium-potassium pump and calcium pump become functionally impaired. High  $iCa^{2+}$  has several vasoconstrictive effects which lead to hypertension, an indirect result of low magnesium status. Dietary calcium is directly proportional to dietary magnesium. Serum magnesium does not reflect true magnesium status as do intracellular magnesium measurements. Several studies on the effect of calcium on blood pressure need these added considerations of magnesium status to fully understand the impact of the Mg:Ca ratio as the primary cause of hypertension and other aspects of Syndrome X. Magnesium supplementation above 15 mmol per day are required to normalize high blood pressure in unmedicated hypertensive patients while 15 mmol per day will lower high blood pressure in patients treated with anti-hypertensive medications. In most humans, healthy



blood pressure depends upon a balance of both Na:K and Mg:Ca ratios at both cellular and whole body levels which, in turn, require adequate, long-term intakes of nutritional magnesium. The knowledge that low magnesium causes imbalance in both cellular and physiological calcium widens our view of the studies showing hypertensives have abnormal calcium metabolism..

Conversely, consuming too much magnesium typically causes diarrhea as the body attempts to excrete the excess. High magnesium foods include dark leafy greens, nuts, seeds, fish, beans, whole grains, avocados, yogurt, bananas, dried fruit, dark chocolate, and more. The current daily value (DV) for magnesium is 400mg.

To be continued....!!

The composition of blood , especially gas exchange and pH stimulates prāna vāyu to change in blood pressure in maintaining homeostasis .. : in presence of circulatory shock , decreased blood flow stimulates prāna vāyu to increase blood pressure to maintain perfusion..: āyusho anuvriti pratyaya bhooto bhavati akupitah... Rakta is essential for Jeevana hence any qualitative or quantitative change in composition of blood or change in circulation influence the action of vāyu and eventually the change in blood pressure occurs.. flow , pressure and resistance , all work with coordination to maintain tissue perfusion..

The carotid bodies are located on the external carotid arteries near their bifurcation with the internal carotids. Each carotid body is a few millimeters in size and has the distinction of having the highest blood flow per tissue weight of any organ in the body. Afferent nerve fibers join with the sinus nerve before entering the glossopharyngeal nerve. Hypoxemia, hypercapnia and acidosis lead to an increase in carotid body receptor firing. When hypoxemia results in a PO<sub>2</sub> lower than about 80 mmHg (threshold PO<sub>2</sub>), receptor firing is stimulated (normal arterial PO<sub>2</sub> is about 95 mmHg). Any elevation of PCO<sub>2</sub> above a normal value of

40 mmHg, or a decrease in pH below 7.4 causes receptor firing. If respiratory activity is not allowed to change during chemoreceptor stimulation (thus removing the influence of lung mechanoreceptors), then chemoreceptor activation causes bradycardia and coronary vasodilation (both via vagal activation) and systemic vasoconstriction (via sympathetic activation). If respiratory activity increases in response to the chemoreceptor reflex, then increased sympathetic activity stimulates both the heart and vasculature to increase arterial pressure. A decrease in carotid body blood flow as can occur during circulatory shock also increases receptor firing.

2 Aug 2014

Hypertension ; the role of salt in HTN ; Excess salt -nonmodulation ( vikriti vighaata abhaava ch.ni.4) - activation of renin-angiotensin -aldosterone system( RAAS)- increased heart rate , salt and water retention ( =increased cardiac output ) , vasoconstriction (increased total peripheral resistance ) -> HTN.. ayurved ; lavana - rakta vridhi - increased CO -HTN..

Therefore salt reduction is helpful in reducing BP..

Anxiety- sympathetic overstimulation -HTN .. kaama , shoka, bhaya - vaata prakopa ( especially praana vaata)- HTN...Therefore yoga is helpful in HTN.. Pomegranates ( Dadim/ anaara/daalimb ) is known as hridya ; it contains antioxodants , polyphenols, fibres rich in punicalagins, potassium , iron , vitamins . These nutrients help to reduce cholesterol , blood pressure, stress and improve digestion , immunity , libido and prevent cancer alzheimer disease.. potassium is vasoactive. it causes vasodilation so reduces BP in salt sensitive hypertensive patients. Its effective in anaemia , hypokalaemia.. its very helpful in familial periodic hypokalaemic paralysis..

31 July 2014

**Papilledema** is caused by intracranial hypertension due to malignant hypertension with renal insufficiency .. so , confirm the level of blood pressure and renal functioning.. its an emergency so anti hypertensive drugs; nicardipine/labetalol/enalaprilat /esmolol/nitroglycerine/phentolamine/hydralazine/nitroprusside are indicated. Initially parenteral therapy is indicated.. later oral therapy to sustain normal blood pressure.. Ayurveda perspective; combination of arjun , punarnava, gokshur, kamal, brahmi,jatamansi, shankhapushpi, pushkarmool..6-12 gm/day in divided dose..Causes of papilledema include brain tumor or abscess, cerebral trauma or hemorrhage, meningitis,encephalitis, cavernous or dural sinus thrombosis ..Treatment as per underlying cause..

In case of resistant hypertension , its essential to confirm " nonadherence to therapy, obesity, alcohol intake, and secondary causes of HTN.. life style modifications like weight reduction , dietary salt reduction , adapt DASH -type dietary pattern, moderation of alcohol consumption and physical activity are indicated.

Ayurveda therapy with virechan ,shirodhara, basti and certain drugs like arjun kamal goksharu punarnava brahmi jatamansi trifala chitrak trikatu vacha chaturjat etc..

25 May 2014

Kaph pitta avaruddhah tu maruto ras moorchchhitah.... hridisthah kurute shoolam uchchhvas avarodhakam param su.utr. 42/132.. Coronary artery atherosclerosis -> myocardial hypoperfusion( ischaemia)-> angina pectoris.....

5 Oct 2014

**Prinzmetal angina** ; Dashamoola pushkarmoola devadaaru erandamoola rasna punarnava trikatu vacha are effective in relieving pain.. vrihatvaatachintamani rasa ,ekangaveera rasa are helpful in decreasing vasospasm.. For arrhythmias prabhakar vati with arjun shatavari brahmi kamal gokshuru.Prinzmetal angina is due to vaata prakopa , so vaataghna drugs( vasodilators) are indicated.. pushkaramoola is choice of drug.. hridabasti can be recommended.. Drugs mentioned above are effective..Prinzmetal's Angina, Variant Angina and Angina Inversa. Unlike typical angina – which is often triggered by exertion or emotional stress - Prinzmetal's angina almost always occurs when a person is at rest, usually between midnight and early morning. These attacks can be very painful.Prinzmetal angina may also be referred to as:Variant angina,vaso spastic angina,prinzmetal's variant angina, angina inversa. Prinzmetal's angina is rare, representing about two out of 100 cases of angina, and usually occurs in younger patients than those who have other kinds of angina.Causes of Variant (Prinzmetal) Angina: the pain from variant angina is caused by a spasm in the coronary arteries (which supply blood to the heart muscle).The coronary arteries can spasm as a result of:Exposure to cold weather,stress,Medicines that tighten or narrow blood vessels,Smoking, cocaine use.

Symptoms of Variant (Prinzmetal) Angina: The pain or discomfort:

Usually occurs while resting and during the night or early morning hours, Are usually severe, Can be relieved by taking medication..

Treatment of Variant Angina | Prinzmetal's Angina

Medicines can help control the spasms. Drugs such as calcium antagonists and nitrates are the mainstays of treatment.The spasms tend to come in cycles – appearing for a time, then going away. After six to 12 months of treatment, doctors may gradually reduce the medication. Avoid cold exposure , stress free life , abandoned smoking are helpful in reducing episodes of chest pain..



7 SEP 2014

**Mitral stenosis(MS)** ;Sanga( loud S1,opening snap, diastolic murmur) - left ventricular inflow obstruction - backward transmission of pressure - vimaarga gamana - left atrial dilatation , AF ,TE - pulmonary venous congestion- pulmonary edema- pranavaha srotodushti - svaasaadi lakshan ( dyspnoea orthopnea PND cough fine crepts etc)- pumonary arterial hypertension(P2 loud) - RVH (para sternal heave )- Rt atrial dilatation , TR (systolic murmur) - systemic venous congestion -rakta vaha srotodushti - raised JVP , congestive hepatomegaly and edema etc.. MS - decreased cardiac output - rasa vaha srotodushti - cold extremity , giddiness , cyanosis , pulsus alternans etc..Treatment of MS ; hridayarnava rasa , chandraprabha vati , arogya vardhini vati , gokshuraadi guggula , punarnavadi kvatha along with arjun shatavari kamala pushkarmoola patola kiratatikta dashamoola haritaki chitraka vacha trikatu.. ( inj Penidure IM at 21 days interval )

24 Feb 2014

Cardiac causes of chest pain ; angina pectoris , angina decubitus , unstable angina , Prinzmetal's variant angina ( vasospastic angina ), acute myocardial infarction , aortic stenosis , aortic regurgitation , cardiomyopathy, acute pericarditis , myocarditis , mitral valve prolapse (rarely) , right ventricular hypertrophy..

Non cardiac causes of chest pain ; Anemia- marked anemia can result in a myocardial O<sub>2</sub> supply -demand mismatch.. Thyrotoxicosis- increased BMR - increase in myocardial demand may result in an O<sub>2</sub> supply-demand mismatch.. Gastroesophageal reflux disease and esophageal spasm can mimic angina ( responsive to NTG).. Biliary colic- gallstones on USG.. Pneumonia- The pain may be pleuritic.. usually visualized on chest X-ray.. Musculoskeletal- costocondritis.... Acharya Charak mentions Urah shool in Vatik gulm , Rajyakshma , kshatkshin ( urahkshat) , kshataj kasaadi.. Acharya Cakrapani on ch.chi. 26/ 101-102. Bhukte adhikam ityadina sannipatik hridroge prayo bhavi shoolam lakshan bheden

vibhajanchikitsati.. Bhukte adhikam ityadina shleshm shoolam uchyate.. Jirne adhikam ityadina anil shoolam , jiryati adhikam iti anen cha paittikam broote.. For differential daignosis between cardiac and noncardiac causes of urah shool ( chest pain ).. The perfect clinical observation of our great Acharya Charak and appropriate interpretation by Acharya Chakrapani.. Namoh...

15 Feb 2014

**Acute pericarditis** ; Chest pain - retrosternaly or left epicardial refered commonly to left neck , shoulder and arm. Chest pain is decreased on siting and lean farward position and increased in supine position. Pericardial rub on left parasternam.. ECG revealed depression of PR- segment , concave elevation of ST-segment in any two standard limb leads and in V2-V6 with reciprocal ST-segment depression in aVR and V1.. in 2nd week ST-segment becomes isoelectric and T wave becomes inverted...

12 Feb 2014

**In tricuspid regurgitation-** backward flow of blood from right ventricle to right atria during systole - right atrial volume overload - increased right atrial pressure - systemic venous congestion :: raised jugular venous pressure , congestive hepatomegaly , systolic hepatic pulsation , +ve hepatojugular reflux , ascites , oedema , soft S1, systolic murmur, S4 , parasternal heave and epigastric beats are present.. Ayurved perspective : Hriday gat dushti - vimarg gaman - rasavah sroto dushti - shoth , pandu . Raktvah sroto dushti - yakriddalyudar , plihodar , jalodar , kamala , siragat raktadhikya.....t/t.. combination of Arjun , punarnava , gokshuru , varun , shatavari. Chandraprabha vati and shwet parpati.. salt restriction..

22 Feb 2014

Indians are suffering from IHD , HTN , DM type-2 , more commonly due to growing incidence of the metabolic syndrome in indian population since

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centuries.. The reason is central adiposity and sedentary lifestyle which in turn develop insulin resistance ; hyperinsulinaemia , glucose intolerance & DM , and hypertension.. Acharya charak mentions " aasyaasukham swapnsukham... in ch.ch.6/4.. with reference to prameh.. and mentions vyaayaam (exercise) as a part of treatment along with cessation of etiological factors ( santarpanoth hetu-fat and energy rich diet ).. Metabolic syndrome ( atisthool - vikaaraan daaroonaan kritva naashayanti ashu jivitam ch.su.21/8 ) is present in indian population since centuries because of earliest civilization in this part of world..

11 Feb 2014

**Aortic regurgitation** -> backward flow of blood from aorta to left ventricle during diastole - increased left ventricular end diastolic volume - volume overload - left ventricular dilatation and eccentric left ventricular hypertrophy : c/f - palpitation , diaphoresis , dyspnoea, orthopnoea , paroxysmal nocturnal dyspnoea, anginal chest pain, especially during night .. jarring of the entire body and bobbing motion of head with each systole , dance like movement in carotid arteries , Corrigan's pulse , Quincke 's pulse , Traube's sign and Duroziez's sign are present.. wide pulse pressure with systolic hypertension and low diastolic blood pressure.. Diastolic thrill , A2 either soft or absent , S4 , high pitched diastolic murmur.. ECG-LVH and LV strain.. t/t diuretics, vasodilators... penicillin therapy if syphilitic aortitis or infective endocarditis.. Ayurved t/t same as in aortic stenosis with gokshuru , varun and shwet parpati like mutral drugs..

3 Feb 2014

**Aortic stenosis**-> left ventricular outflow obstruction - left ventricular pressure overload - concentric hypertrophy of lv- dyspnea on exertion , angina pectoris and exertional syncope:: slow rising pulse, decreased pulse pressure , systolic thrill , ejection sound , mid systolic murmur , loud A2 and S4.... in ECG LVH... Ayurved aspects -- sang- dhatukshaya- vat prakop - shwas, urahshool and murchchha.. and abnormal heart sounds ( prakritih sparsh shabdayoh ch.su 12/8).... Treatment :

combination of dashmool pushkarmool punarnava arjun kamal haritaki brahmi chitrak vacha and shatavari with hridayarnav ras and prabhakar vati... modern t/t beta blockers ACE inhibitors nitrates and statins..

Aortic stenosis-> pressure overload-> increased systolic pressure->wall thickening->concentric hypertrophy... Aortic regurgitation ->volume overload-> increased diastolic pressure-> chamber enlargement-> eccentric hypertrophy..

29 Jan 2014

In PMBV ( percutaneous mitral balloon valvotomy ), a catheter is directed into left atrium after transseptal puncture, and a single balloon is directed across the valve and inflated in the valvular orifice . PMBV is indicated in patient of mitral stenosis with mitral valve area less than 1.5 cm<sup>2</sup>.

27 Jan 2014

**Patent ductus arteriosus** ;; in adults who were born with a large left-to-right shunt through the ductus arteriosus, pulmonary vascular obstruction ( Eisenmenger syndrome) with pulmonary hypertension, right-to-left shunting and the toes-but not the fingers- become cyanotic and clubbed, a finding termed differential cyanosis. Continuous "machinery" murmurs and characteristic thrill are present....

19 Jan 2014

The electrocardiogram (ECG or EKG) is a graphic presentation of electric potentials generated by the heart. The signals are detected by metal electrodes attached to the extremities and chest wall and are amplified and recorded by mechanical device known as electrocardiograph. ECG leads actually display the instantaneous differences in potential between these electrodes. Action potential



is depolarization(cardiac contraction ) and repolarization(cardiac relaxation) and presents waves , complexes and segments on graph paper. Prayatn of udaan vaat is action potential and bal is for strength of contractility of myocardium. Controlling of rate and rhythmicity by praan vaat(ANS-autonomic nervous system). Cardiac perfusion ,preload and afterload by vyan vaat.. Enzymatic activities in cardiac cells by samaan vaat. The excretion of intermediate and end products of metabolism is done by apaan vaat.. The abnormalities of all these functions are detected by ECG...

14 Jan 2014

WPW syndrome, is also known as preexcitation syndrome,is associated with AV bypass tracts , commonly present in Ebstein's anamoly. ECG reveals a short PR interval (<.12sec), a slurred upstroke of QRS complex(delta wave), and a wide QRS complex (>\_.12sec). Paroxysmal supraventricular tachycardia, atrial flutter , atrial fibrillation and vetricular fibrillation may occur as complications. C/f are fatigue, dyspnoea, palpitation, angina,syncope and heart failure.. t/t - beta blockers or calcium channel blockers. As per ayurved perspective vitiated vaat is cause of avyaahatgati which accelerates velocity of conduction of impulse to ventricles and leads to preexcitation.. t/t prabhakar vati, arjun , shatavari and brahmi or shalparni with milk as mentioned by charak for hriday gat vaat( ch.ch.28/96). Bradyarrhythmias and tachyarrhythmias can be considered as hriday gat vaat..

13 Jan 2014

ECG findings ; PR-Interval is prolonged (>0.20sec) in acute rheumatic fever and shortened (<0.12sec) in wolf-parkinson-white (wpw) syndrome...

19 Jun 2013

Tridoshaj hridrogi+ nishevate til kshir gudaadeeni->rasdoshkritah->granthitasyopajaayate->hridayekadeshe sankledam->sankledat->krimayah

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ashya bhavanti... ch.su.17/36-38.. Group A beta-hemolytic streptococci+ dysfunctional immune response-> molecular mimicry between group specific carbohydrate of group A streptococcus and glycoprotein of heart->rheumatic vulvulitis-> rheumatic heart disease .

3 June 2014

Tadev darbh mriditam raktam bastim pradapayet CH.SI.6/83 Here, darbh is used as anti coagulant. on experimental animal model it is found effective similar to heparin. Great observation by acharya charak;we can use darbh as anti coagulant in venous thrombosis, thrombo-embolism, disseminated intra-vascular coagulation etc.

5 gram darbh (not durva) is found effective to prevent clotting of 50 ml blood and 10 gram to prevent clotting of 100 ml of blood.. it means 1:10 ratio (darbh : blood) is to be used before administering through basti...

Darbh->anti-coagulant mriditam raktam ; like heparinized blood.. so great scientific observation and effective therapeutic use by all time great acharya charak... pratahkalin sadar pranam maharshi...

Try to administer darbh mixed blood of animal through basti in leukemia.... darbha mriditam raktam bastim pradaapayet.. ch.si.6/83..

कफानुबन्धे रुधिरे सपित्ते कण्ठागते स्यात् ग्रथिते प्रयोगः।

युक्तस्य युक्त्या मधुसर्पिषोश्च क्षारस्य चैवोत्पलनालजस्य॥

-(च.चि.५\९३)

Kshara of Utpala naala ( a species of lotus) is found effective as thrombolytic (fabrinolytic) drug.

sakshaudram grathite rakte lihyat paravatam shakrit ch.chi.4/72 paravat shakrit, means fecal matter of pigeon, is anti-thrombotic and thrombolytic drug.

12 Feb

**According to Caraka Samhita, plants termed as hridya ; effective in cardiovascular diseases are as follows**

Botanical name/Family

Common names

Useful parts

Magnifera indica (Anacardiaceae)

Aam, Amba

Fruit

Gmelina asiatica, (Verbenaceae)

Badhar

Fruit

Carissa carandas, (Apocynaceae)

Karanda

Fruit, Bark, Leaves

*Garcinia indica*

(Guttiferae)

Amsul

Fruit

*Garcinia pedunculata*

(Guttiferae)

Amlavetta

Fruit

*Ziziphus jujuba*

*Ziziphus nummularia*

(Rhamnaceae)

Kuval, Bor

Fruit, Leaves

*Punica Granatum*

(Lythraceae)



Anardana

Flowers, Fruits

Citrus medica Linn. (Rutaceae)

Mahalung

Fruit

Bibhitaki, Kachur, Nagarangaphala,

yavani, arjaka, shigru, shaleyā from the group of leafy vegetables

Shakara, pakvarasa, madhvasava, madhvikasava, sauviraka, tusodaka from medicated alcohol group

Navaneet, manda, sauvarchala, ragashadava, godhuma, sunthi, yavakshara are from the food accessories group.

Yava

Chataka egg and flesh

Khajur (Dates)

Utpal

Lavaliphala

8 April 2016

The scientific correlation ; acharya charak , ch.soo 27 ; katuko raso shonita sanghaatam bhinatti , modern research reveal that black Piper is antithrombotic drug. Kinchit amlam bhinatti cha ; acharya charak .. Pomegranate fruits consist of antithrombotic , anticoagulant and thrombolytic properties ( contemporary science observation ).. अहिततम् - गोमांसं मृगमांसानाम् , च.सू.25 , cow / beef / red meat - induces hyperlipidaemia , obesity , DM , CVD , Alzheimer's disease , arthritis , colon cancer , mad cow's disease , etc .. Rabbit meat is , शशः स्वादुः प्रशस्तः च संनिपाते अनिलावरे । मधुरा मधुराः पाके त्रिदोषशमनाः शिवाः । च.सू. 27/77 , so healthy and lean that doctors prescribe rabbit meat diets to people who are overweight and obese. Because the fat and calorie levels are so low, but protein so high, one can radically change their life by eating arabbit meat diet and exercising.

## **Modern science and ayurved perspective in cough /kāsa**

Cough Reflex: The bronchi and trachea are so sensitive to light touch that very slight amount of foreign matter or other causes of irritation initiate the cough reflex. The larynx and carina (the point where the trachea divides into the bronchi) are especially sensitive, and the terminal bronchioles and even the alveoli are sensitive to corrosive chemical stimuli such as sulfur dioxide gas or chlorine gas. Afferent nerve impulses pass from the respiratory passages mainly through the vagus nerves to the medulla of the brain. There, an automatic sequence of events is triggered by the neuronal circuits of the medulla, causing the following effect. First, up to 2.5 liters of air are rapidly inspired. Second, the epiglottis closes, and the vocal cords shut tightly to entrap the air within the lungs. Third, the abdominal muscles contract forcefully, pushing against the diaphragm while other expiratory muscles, such as the internal intercostals, also contract forcefully. Consequently, the pressure in the lungs rises rapidly to as much as 100 mm Hg or more. Fourth, the vocal cords and the epiglottis suddenly open widely, so that air under this high pressure in the lungs explodes outward. Indeed, sometimes this air is expelled at velocities ranging from 75 to 100 miles per hour. Importantly, the strong compression of the lungs collapses the bronchi and trachea by causing their non-cartilaginous parts to invaginate inward, so that the exploding air actually passes through bronchial and tracheal slits. The rapidly moving air usually carries with it any foreign matter that is present in the bronchi or trachea. The above explanation holds true as a physiological as well as a disease process.

The main pathogenesis has 5 steps viz (i) pratihatō vayu i.e obstruction or avarodh to the normal movement of vayu. It may occur due to the bronchospasm or due to mucus or due to tumour or any foreign body. As said by Chakrapani that kapha (mucus) etc are the cause for the obstruction to the natural movement of the vata.

(ii) urdhwa srota samasrita: this step explains the complete process of the afferent nerves taking the impulse to the cough centre leading to the epiglottis closure and shutting down of the vocal cords to entrap the air within the lungs.

(iii) udānabhāvamāpannaḥ: third and the important step is to gain bala to the urdhwa gati which is brought about by the contracture of abdominal muscle pushing against the diaphragm while other expiratory muscles, such as the internal intercostals, also contract forcefully.

(iv) khāni sarvāṇi pratipūrayan i.e. the pressure in the lungs rises rapidly. Khāni means srotas i.e air gets filled up in the complete pulmonary passage.

(v) vāyōḥ saramhasaḥ: Chakrapani comments on saramhasa as sa vegasya i.e. with full speed. The vocal cords and the epiglottis suddenly open widely, so that air under this high pressure in the lungs explodes outward. Secondly, the strong compression of the lungs collapses the bronchi and trachea by causing their noncartilaginous parts to invaginate inward, so that the exploding air actually passes through bronchial and tracheal slits.

The specificity of the sound depends on the obstruction caused to the movement of vayu (pratighāta viśēṣeṇa). The pratighātaviśēṣeṇa may be due to consolidation as seen in pneumonia, tuberculosis etc. Obstruction may also be caused due adenocarcinoma or by simple process of bronchospasm. The presentation on auscultation like fine crepts or rhonchii is nothing but the pratighāta viśēṣa which helps in diagnosis of the cardiorespiratory diseases

#### **My approach to charak chikitsa sthaana 18/ 10-13 :**

rūkṣa, śīta, kaṣāya, alpa, pramitānaśan, striyaḥ vēgadhāraṇam, āyāsō are the triggering factors (pravartaka) for vātaj kāsa.



rūkṣata brings dryness in the tract or in other words reduce the mucosal secretion (kaphaṁ śuṣkaṁ), sita and kashaya rasa are known to have constricting effect whereas alpa, pramitanasan and stri atisevan causes reduce nutrition of the dhatu. Vegadharan especially adhovega i.e mala, mutra, purisa and apan vata helps the apan vayu to get udan bhav apanna as upward movement of apan vata takes place i.e. diaphragmatic movement is increased as discussed above. Further excessive exercise increases the breathing rate and also has impact on the ciliary movement of the respiratory tract.

All the respiratory passages, from the nose to the terminal bronchioles, are kept moist by a layer of mucus that coats the entire surface. The mucus is secreted partly by individual mucous goblet cells in the epithelial lining of the passages and partly by small submucosal glands. In addition to keeping the surfaces moist, the mucus traps small particles out of the inspired air and keeps most of these from ever reaching the alveoli. The mucus itself is removed from the passages in the following manner. The entire surface of the respiratory passages, both in the nose and in the lower passages down as far as the terminal bronchioles, is lined with ciliated epithelium, with about 200 cilia on each epithelial cell. These cilia beat continually at a rate of 10 to 20 times per second and the direction of their "power stroke" is always toward the pharynx. That is, the cilia in the lungs beat upward, whereas those in the nose beat downward. This continual beating causes the coat of mucus to flow slowly, at a velocity of a few millimeters per minute, toward the pharynx. Then the mucus and its entrapped particles are either swallowed or coughed to the exterior. In vataj kasa the repeated exposures of triggering factors either disturbs the ciliary movement or reduces the mucosal secretion causing the respiratory tract to continuous exposure to irritants. Thus a protective mechanism of cough is initiated in the form of cough but when it continues for a long time it is considered as a disease. This holds true for the phenomenon of allergic cough.

Due to reduce mucosal secretion either there is dry cough (śuṣka kāsa) or after repeated coughing little mucus is removed with difficulty (kṛcchrānmuktvā'lpatāṁ vrajēt).

Several substances formed in the lungs themselves often quite active in causing bronchiolar constriction. Two of the most important of these are histamine and slow reactive substance of anaphylaxis. Both of these are released in the lung tissues by mast cells during allergic reactions, especially those caused by pollen in the air. Therefore, they play key roles in causing the airway obstruction that occurs in allergic asthma; this is especially true of the slow reactive substance of anaphylaxis. The same irritants that cause parasympathetic constrictor reflexes of the airways—smoke, dust, sulfur dioxide, and some of the acidic elements in smog—often act directly on the lung tissues to initiate local, non nervous reactions that cause obstructive constriction of the airways.

Pratāmyati means the blackout which is observed in cough syncope.

Spontaneous subconjunctival haemorrhages are the result of the rupture of small vessels from increased intravascular pressure, during the explosive and repetitive bouts of coughing associated with whooping cough and in patients with hypertension..

Rajasā dhūmavātābhyāṁ ;

### **The important etiological factors of asthma and COPD :**

Air pollutants, such as sulfur dioxide, ozone, and diesel particulates, may trigger asthma symptoms. Indoor air pollution may be more important with exposure to nitrogen oxides from cooking stoves and exposure to passive cigarette smoke. There is some evidence that maternal smoking is a risk factor for asthma, but it is difficult to dissociate this association from an increased risk of respiratory infections.

Allergen: The increase in house dust mites in centrally heated poorly ventilated homes with fitted carpets has been implicated in the increasing prevalence of asthma in affluent countries. Domestic pets, particularly cats, have also been associated with allergic sensitization, but early exposure to cats in the home may be protective through the induction of tolerance. Pollens usually cause allergic rhinitis rather than asthma, but in thunderstorms, the pollen grains are disrupted and the particles that may be released can trigger severe asthma exacerbations (thunderstorm asthma).

Occupational Exposure Occupational asthma is relatively common and may affect up to 10% of young adults. Occupational asthma may be suspected when symptoms improve during weekends and holidays.

Dutch hypothesis. This suggests that asthma, chronic bronchitis, and emphysema are variations of the same basic disease, which is modulated by environmental and genetic factors to produce these pathologically distinct entities. The alternative British hypothesis contends that asthma and COPD are fundamentally different diseases: Asthma is viewed as largely an allergic phenomenon, whereas COPD results from smoking-related inflammation and damage. Rajasā dhūmavātābhyām explains the approach of Caraka which explains both the hypothesis wherein allergic and inflammatory and damage factors have been explained together.

It may also be noted that Intrinsic Asthma: A minority of asthmatic patients (approximately 10%) have negative skin tests to common inhalant allergens and normal serum concentrations of IgE.

24 March 2013

On charak sidhisthan-9/4 acharya chakrapani stated that although the place of apan is medhradi, but it may be even hriday, because- hridayaavyatiriktaanuvidhayitvat hridayaashrit uchyate.. Hriday plays a role in circulation of rasaadi ambu dhatu, which constitute not only dhatu sarbhag but

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also kittabhag,through vyan vat karm.. vyanen rasdhaturhi vikshepochitkarmana..ch.chi15/36- but as apan is responsible to eliminate kitta,it is apan which transports the kitta to be excreted through orifices.. This is reason why acharya charak mentions hridgadaha in saman avrit apan(ch.ch.28/205

### **The interlinke between pratishyāya and shwāsa roga ;**

Raja, dhuma, excessive sexual intercourse, ajirna, shita ambu are some common aetiological factors between pratishyaya and hikka shwas which explains that this factors have impact on local immunity of pranvaha srotas. As previously discuss in hikka shwas adhyaya this factors stimulate the immune response leading to secretion from the nasal cavity (pratiśyāyamudīrayēttu). Śītairavaśyayā has been mentioned as one of the cause wherein avashyaya means tushaar i.e. water drops which may be compared with dew drops, fountains or in present day condition coolers and air conditioners which have been found to be common aetiological factors even in present era. Ch.chi 26/ (104-105)..

Simons reviewed some of the early concepts linking Allergic Rhinitis ( AR ,) and asthma, and suggested that the connection between the two had actually been known for centuries, but that specialization in medicine, as well as in medications, led to the perspective of separate disease entities. To reconnect asthma and AR, Simons proposed using the term 'allergic rhinobronchitis'. Another current concept is 'one airway, one disease', which was originally suggested by Grossman in 1997 . It seems logical that an uninterrupted air passage from the nose to the alveolar ducts of the lungs would have many similarities. The following discussion elaborates on the connections between AR and asthma in terms of their epidemiology, pathophysiology and similar responses to treatment. Each of these disorders has a variety of effective treatment modalities; asthma responds well to inhaled corticosteroids and beta2-agonists, as well as to a new adjunctive therapy approved in Canada, the anti-immunoglobulin E (anti-IgE) agent omalizumab . Treatment for AR usually involves various forms of antihistamines and decongestants, as well as glucocorticosteroids ..



21 Aug 2014

Pratishyaaya/Rhinitis ;Tanu sraava or jalaabhah sraava is present in vaatika pratishyaaya , indicates vaata prakopa occurs through its rooksha guna which leads to vasodilatation and in turn watery nasal secretion.. causes are noninfective , predominantly allergens ( kshavathu/sneezing is predominantly present in allergic rhinitis ).. Allergens interplay with IgE on mast cells and subsequently histamine mediated vasodilatation occurs.. one an other example in paittika kaasa when there is tanu kaphe, snigdha sheeta chikitsa and in ghane kaphe , rooksha sheeta chikitsa are indicated by acharya charak ( ch.chi 18/85-86).. snigdha chikitsa for tanu kaphe indicates that alpa kapha is present so constituency of kapha becomes less.. and rooksha chikitsa is to decrease constituency of kapha in ghane kaphe.. it shows the role of rookshta in inducing tanuta and snigdhatata in ghanata , so tanu shraava in vaatika and ghana sraava in kaphaj pratishyaaya are present (ch.chi 26/105;106)..only hence Hareetaki yukta Agasthya Rasayana plays a good role in providing relief in ac.Rhinitis, where in tanu srava is present, due to vata predominance.And Kapooradi churnam gives good relief in kaphaja kaasa with snighda mucous. vyaaghri haritaki avaleha is effective in vaatika pratishyaaya , vaatika kaasa and tamaka shvaasa..

28 March 2015

**Acharya charak and modern hypothesis of Bronchial asthma and COPD ( Shvaasa roga ) ;**

the considerable overlap between persons with asthma and those with COPD on airway responsiveness ,airflow obstruction , and pulmonary symptoms led to the formulation of Dutch hypothesis. This suggests that asthma , chronic bronchitis and emphysema are variations of the same basic disease, which is modulated by environmental and genetic factors to produce these pathologically distinct entities. The alternative British hypothesis contends the asthma and COPD are

fundamentally different diseases ; Asthma is viewed as largely an allergic phenomenon, while COPD results from smoking-related inflammation and damage. Acharya Charak mentioned Rajasa (pollen grains/allergens) Dhooma ( smoking ) vaataabhyam.... etc as hetu of shvaasa roga.. as per acharya chakrapani ; rajasa ityaadinaa prayo vaata prakopakagano vichchhidyoktah gadaavimaavityantena , nishpaava ityaadinaa kaphakaaranatayaa hikka shvaasayoh kaphaprakopa hetu gano abhihitah ; tadanena vaatajanaka kaphajanaka hetu varga dvaya vichchhet paathena vaatakaphayoh atra svahetukupitatvena svaatantryam darshayati , na anubandhamaroopatvam.. it means two different groups of etiological factors induce shvaasa roga separately ; vaata and kapha are vitiated by their hetu separately to initiate disease process.. Both extrinsic and intrinsic factors are mentioned as hetu of shvaasa roga. In samprapti of shvaasa roga acharya charak mentions vishvagvrajati ( sarvato gachchhati ) , means nonuniform ventilation , discarded ventilation and mismatching between ventilation and perfusion , a cardinal pathophysiology of shvaasa roga.. in an other reference ( ch.chi 18/131) acharya refered Tamakah kaphakaase tu syaachchet pitta-anubandhajah... when there is secondary infection occurs in patient with chronic bronchitis , airway obstruction develops and in turn manifests severe dyspnea similar to episode of bronchial asthma (earlier known as infective bronchial asthma ). Since centuries the concept about shvaasa roga is very clear in ayurveda.. Determination of the validity of Dutch hypothesis Vs. British hypothesis awiats identification of the genetic predisposing factors for asthma and /or COPD ,as well as the interactions between these postulated genetic factors and environmental risk factors.. acharya charak mentioned very clearly the role of vaata and kapha in shvaasa roga ; Yadaa srotaansi sanroodhya maarootah kaphapoorvakah. Vishvagvrajati sanroodhah tadaa shvaasaat karoti sah..maaroota , kapha →Hyperresponsiveness of bronchial smooth muscles and inflammation of airways →airway obstruction →shvaasa roga ( bronchial asthma and COPD)..Acharya charak mentions tila taila as hetu of hikkashvaasa ( ch.chi 17).. Nishpaava maasha pinyaaka tila taila nishhevanaat are observed as hetu of pandu

roga and shvaasa hikka.. Acharya charak mentioned the same hetu in vidhi shonatiya adhyaaya.. means these are causes of raktaja roga. I think these all nishpaavaadi are hetu of kapha prakopa and rakta dushti..its kaphaprakopaka hetu , very clearly told by acharya chakrapani..Tila may lead to histamine liberation by mast cells after allergen - IgE binding on surface of mast cells.. Hyperresponsiveness may be caused by tila or tila tail or any hetu mentioned in shvaasa roga.. le srotaansi sanroodhya/airway obstruction.. references ; Charak sanhita and Harrison 's principles of internal medicine...

8 Sept 2014

Complications of kaasa --Kaasaat shvaasa kshayah chhardi svarasaadaadayo gadah , bhavanti upekshayaa.. A.H. ni 3/38.. chronic bronchitis (Kaasa) --COPD (Dyspnea)-; Pulmonary hypertension ( hemoptysis ) -; Rt heart failure (cyanosis, edema )..Blue bloater + Sec. Infection by mycobacterium tuberculosis -; pulmonary Tuberculosis ( kshadarooa or ekaadasha roopa) -: laryngeal TB...

Acharya charak ; kaasam aatyayikam matvaa kshatajam tvarayaa jayet ( ch.chi 18/134).. kshataj kaasa -: massive hemoptysis -: acute circulatory failure , an emergency need prompt blood transfusion to maintain blood volume.. acharya charak ; madhuraih jeevaneeyaih cha bala maansa vivardhanaih ( ch.chi 18/134).. sadaaham kaasino raktam shthivatah (hemoptysis) sabale anile (140)...



30 May 2015

**The conceptual study of connective tissue diseases as one of causative factors in Interstitial lung diseases (ILDs) : both modern and ayurveda perspective ;**

Patients with ILDs come with the onset of progressive exertional dyspnea or a persistent nonproductive cough. Hemoptysis , wheezing and chest pain may be present. Often , the identification of interstitial opacities on chest X-ray focuses the diagnostic approach on one of the ILDs. Rheumatoid arthritis is one of connective and autoimmune diseases which cause ILDs.. inflammation in the air space and alveolar walls and interstitial fibrosis are present due to autoimmunity.. the presence of jts pain , stiffness and deformity like swan neck etc with +ve antinuclear antibodies and anti- immunoglobulin antibodies ( RA factors ) confirm the diagnosis , however these tests are +ve even in absence of defined connective tissue diseases. ILDs are characterized as a significant part of a multiorgan process , as may occur in the connective tissue diseases ( SLE, RA , Ankylosing Spondylitis, systemic sclerosis , sjogren's syndrome , polymyositis-dermatomyositis. Aamavaata mentioned in madhav nidan is classical example of autoimmune diseases , otherwise in relation to grahani chikitsa acharya charak mentions yakshma peenas mehaadeen kaphajaan kaphasangatam; aamavisha along with kapha when reaches into urahsthaana , can induce yakshmaa peenasa like diseases. I consider the role of aamavisha yukta kapha as part of autoimmunity , since vaata is major initiator of immune phenomenon , so vaata and kapha as predominant dosh can be considered in ILDs. The presence of inflammation and fibrosis is due to autoimmune reaction , ie aamaavisha yukta kapha reactions with vaata on surface of epithelium.. shirisha tulasi gorakhamundi darvi haridra amrita karkatashringi shunthi pippali aamalaki dashamoola pushkaramoola kantakaari bharangimoola kushtha ashwagandha like drugs are found somewhat effective in ILDs...Salient features in pathogenesis of ILDs ; the lung is naturally exposed to repetitive injury from a variety of



exogenous and endogenous stimuli . Several local and systemic factors e.g. fibroblasts , circulating fibrocytes, chemokines , growth factors , and clotting factors contribute to tissue healing and functional recovery. Dysregulation of this intricate network through genetic predisposition and autoimmune conditions ,or superimposed diseases can lead to aberrant wound healing , with the result of pulmonary fibrosis. Alternatively , excessive injury to the lung may overwhelm even intact reparative mechanisms and lead to pulmonary fibrosis..For inhibition of dysregulation , or promotion of reparative mechanisms , rasaayana chikitsa may be helpful.. Tulasi saarivaa kushtha haridraa daruharidra ashwagandhaa gokshuru shati lashuna jivanti musta bhumyamalaki pippali dashamoola kutaki haritaki aamalaki tejapatra shirisha bilva pushkaramoola chitraka pravaala tamra lauha rajata bhasma abhraka etc can be tried in different form..

4 Feb 2016

**Salient features of shvaasa roga & Hridroga** ; Rajasa is told as first hetu in shvaasa roga , as per acharya chakrapani , Rajasa is in vaata prakopaka gana . pollen grains/ allergens react with Mast cells, dendritic cells and in turn activates TH2 cells & eosinophil , neutrophil (inflammatory cells ). There is secretion of inflammatory mediators (histamines, leukotriens, PAF , etc ) , which lead to bronchospasm, plasma exudation, mucus secretion , etc due to underlying inflammation.. Asthma may be regarded as a disease with continuous inflammation and repair proceeding simultaneously..Please note one thing that without pitta there may be inflammation. Best example ; in abhighaataja jvara , vaata prakopaka and rakta dushti are present which manifest jvara , shotha , vaivarnya and vedanaa , similar to features of inflammation.So , if allergens are triggering factors , they will work as vaata prakopaka hetu and in turn develop inflammatory response , hypersensitivity of bronchial smooth muscles and manifest dyspnea , wheezing cough , as sine qua nan of bronchial asthma.. Histamines and related factors are synthesised and secreted by interplay between triggering and genetic factors.. And there is too many triggering factors , can be

classified as vaata , pitta and kapha prakopaka .Earlier , researchers found the role of vasoactive intestinal peptides as an intrinsic factors to play role in pathogenesis of bronchial asthma. Histamines and its receptors are major inflammatory agents, H1, H2, H3 histamines receptors are isolated.. H2 receptors are found as gastric acid stimulants.. Aamaashaya is told origin of shvaasa roga , considering stomach and upper intestine , the correlation looks logic.. After virechan these peptides may be excreted out , further research is needful to reach on any accepted hypothesis...

As per Dutch hypothesis , bronchial asthma , chronic bronchitis , and emphysema are variations of the same basic disease , I found the same approach while studying charakokta shvaasa roga.. Shvaasa roga -praanavasa sroto dushti - hridaya dushti - rasavaha srotodushti..-rasaadi dhaatunaam upashoshana (aruchi , paandu , kaamala , shothaadi ) . COPD -Pulmonary hypertension -cor pulmonale - systemic venous congestion -raised jvp , congestive hepatomegaly ( jaundice, indigestion, ascites etc), edema.. Marma upaghaata - hridaya upaghaata - shotha.. Acharya chakrapani ; marmopaghaata iha doshakrita Eva geyah..

Gadaatichaara ( iti rogaanaam asamyaka upachaara ; shvaasa rogasya asamyaka upachaara ) - hridroga.. (Ch.chi.26). Vaivarnya in hridroga can be seen as cyanosis, pallor , icterus , splinter haemorrhage , etc. Moorchchha in hridroga due to Aortic stenosis , obstructive cardiomyopathy , hypertension , acute coronary syndrome , arrhythmia , thromboembolism ,etc..

#### **Ch.ch.17/17 ;**

uraḥsthaḥ kaphamuddhūya: Explains the hyperplasia of mucus secretion as seen in COPD wherein small airways may become narrowed by cells (hyperplasia and accumulation), mucus, and fibrosis. Characteristic cellular changes include goblet cell metaplasia, with these mucus-secreting cells replacing surfactant-secreting Clara cells. Smooth-muscle hypertrophy (kupyati maruta) may also be present.

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These abnormalities may cause luminal narrowing by fibrosis, excess mucus, edema, and cellular infiltration.

Vitiated vata expels kapha from its sthana can be easily understood in case of cystic fibrosis. Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) which is an integral membrane protein that functions as an epithelial anion channel. The ~1480-amino-acid molecule encodes a passive conduit for chloride and bicarbonate transport across plasma membranes of epithelial tissues, with direction of ion flow dependent on the electrochemical driving force. The function resembles that of vata thus it can be said that CFTR is vatadharmi dravya. Along respiratory mucosa, CFTR is necessary to provide sufficient depth of the periciliary fluid layer (PCL), allowing normal ciliary extension and mucociliary transport (normal function of vata). But in cystic fibrosis this CFTR-deficient airway cells exhibit depleted PCL (kupita vata), causing ciliary collapse and failure to clear overlying mucus. In airway submucosal glands, CFTR is highly expressed in acini and may participate both in the formation of mucus and extrusion of glandular secretion onto the airway surface (udhuya kapham) thus obstructing, the gati of pran leading to oxygen and carbon dioxide mismatch and therefore difficulty in breathing.

In other condition this kapha (inspissated mucus) helps in further growth of bacterial infection leading to collateral tissue injury and further aggravates respiratory decline.

Sometimes inorganic and organic dust, are associated with chronic mucus hypersecretion (chronic bronchitis), with or without reduction of expiratory flow rates.

Mucus hypersecretion Increased mucus secretion contributes to the viscid mucous plugs that occlude asthmatic airways, particularly in fatal asthma. There is hyperplasia of submucosal glands that are confined to large airways and of



increased numbers of epithelial goblet cells. IL-13 (kupyati maruta dharma dravya) induces mucus hypersecretion in experimental models of asthma.

**Inflammatory Mediators** Multiple inflammatory mediators have been implicated in asthma, and they may have a variety of effects on the airways that account for the pathologic features of asthma. Mediators such as histamine, prostaglandin D<sub>2</sub>, and cysteinyl-leukotrienes contract airway smooth muscle, increase microvascular leakage, increase airway mucus secretion (kapham udhuya), and attract other inflammatory cells. Because each mediator has many effects, the role of individual mediators in the pathophysiology of asthma is not yet clear. Although the multiplicity of mediators makes it unlikely that preventing the synthesis or action of a single mediator will have a major impact in clinical asthma, recent clinical studies with antileukotrienes suggest that cysteinyl-leukotrienes have clinically important effects. The mediators are the vata dharma dravya.

#### **Modern time interpretation of ch.chi.17/21-29.**

A hiccup is an involuntary contraction (myoclonic jerk) of the diaphragm that may repeat several times per minute. In medicine, it is known as synchronous diaphragmatic flutter (SDF), or singultus. The hiccup is an involuntary action involving a reflex arc. Once triggered, the reflex causes a strong contraction of the diaphragm followed about 0.25 seconds later by closure of the vocal cords, which results in the classic "hic" sound.

Hiccups are a common experience, and warrant treatment only when they become persistent and bothersome. If persistent, they can affect conversation, concentration, and oral intake, and can lead to frustration, fatigue, and insomnia. They might contribute to an increase in pain. Hiccups are said to be persistent if they last more than 48 hours, and intractable if they last more than a month.



Prana and Pranavahasrotas, udakvahasrotas and annavahasrotas are the one involved in pathogenesis of hikka.

Prana vayu sthana has been mentioned as shira, ura and kantha nasika. Thus dearrangement of pranvayu leads to diseases like hikka and shwas.

Srotodushti leads to vitiation of sthaniya dosha. Clinical case reports mention that lesions of the medulla that involve the area slightly ventral and lateral to nucleus and tractus solitarius cause hiccups. One (of several) explanations for this finding is that such a lesion "irritates" descending information from nucleus solitarius to the phrenic nucleus. The phrenic nucleus consists of a functionally related group of cell bodies in the ventral horn from C3-C5. Axons arising from the phrenic nucleus comprise the phrenic nerve, which innervates the diaphragm. The hiccups result from spasmodic lowering of the diaphragm that causes a short, sharp inspiratory cough. Brain stem lesions involving the area ventral and lateral to nucleus and tractus solitarius result in hiccup.

A condition irritating the vagus nerve such as goitre, pharyngitis or meningitis or a psychological reaction such as shock, fear, grief, excitement, or stress. Even respiratory conditions such as asthma, pneumonia or pleurisy can lead to hiccup.

Cerebral Vascular Accidents: Brain ischemia or stroke is not rare among the individuals with intractable hiccup. If a correct diagnosis is made, intractable hiccup may resolve after initiation of anticoagulant therapy. In addition, hiccup did occur in the patient with systemic lupus erythematosus related to medulla infarct, which was successfully treated with corticosteroid. Steroid was also useful in a patient with systemic lupus erythematosus and hiccup related to vasogenic edema due to aseptic meningitis confirmed on imaging. Briefly, clinicians should consider the probability of CNS ischemia/ stroke among aged people or subjects with vascular disease presenting with persistent or intractable hiccup to avoid delay in diagnosis and initiation of ineffective treatment.

Central Nervous System Tumors, Injury and Inflammatory Diseases

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There is no doubt that any space occupying lesion in the brain has the chance to elicit hiccup. The reported brain tumors to induce serious hiccups included astrocytoma, cavernoma and brain stem tumors etc. Therefore, these hiccups usually resolved after surgical resection of the brain lesions. Cerebellar artery aneurysm also reported to result in hiccup and that was effectively treated after surgery. Brain injury may cause intractable hiccup. It is apparent that a carefully taken history regarding CNS is very important when one encounter these subjects with persistent or intractable hiccup. Apart from other brain stem symptoms like nausea and vomiting, neuromyelitis optica may cause intractable hiccup because this disease is an inflammatory disease involving optic nerves and spinal cord.

#### Lesions along the Peripheral Neuro-pathways

Mediastinal lymph node sarcoidosis, which was likely the lesion invading the hiccup reflex arc, had persistent hiccup that did not respond to steroid therapy. A tumor infiltration on the diaphragm was considered as the obvious cause of intractable hiccup. A patient with herpes zoster involving cranial nerve neurons including the vagus nerve presented with intractable hiccup and diaphragmatic paralysis; acyclovir treatment effectively ameliorated hiccup in this patient.

#### Annavahasrotas

Hiccup may be a physiological phenomenon as in for clearance of air from stomach. A recent explanation by Howes in 2012 suggests that hiccups may have evolved along with other reflexes developed in mammals that allow them to coordinate suckling milk and breathing. Hiccups are only found in mammals, and are most common in infants. This may suggest that they evolved to allow air trapped in the stomach of suckling infants to escape, allowing more milk to be ingested. The hypothesis suggests that the air bubble in the stomach stimulates the sensory limb of the reflex at receptors in the stomach, esophagus and along the diaphragm. This triggers the hiccup, which creates suction in the chest, pulling air from the stomach up and out through the mouth, effectively burping the

animal. This theory is supported by the strong tendency for infants to get hiccups, the component of the reflex that suppresses peristalsis in the esophagus, and the existence of hiccups only in milk-drinking mammals.

Annavahasrotas when vitiated can be the cause of hiccup. Following condition related to Gastrointestinal tract may be cause for hiccup.

gastrointestinal conditions – such as gastro-esophageal reflux disease (GERD), inflammatory bowel disease (IBD) or a small bowel obstruction

conditions involving metabolism – such as diabetes, hypoglycaemia or hyperglycaemia

eating too quickly, eating or drinking too much

Eating too much (fatty or spicy foods, in particular) or drinking too much (carbonated beverages or alcohol) can distend the stomach and irritate the diaphragm, which can cause hiccups.

Udakvahasrotas as discussed later in trishna adhyaya is the one which maintain the fluid balance or in other words electrolyte balance in the body. Disturbance in electrolyte balance such as hyponatremia, hypokalemia, hypocalcemia, and hyperglycemia can lead to hiccups.

Hiccups are observed in many patients with hyponatraemia. Study done by George J, Thomas K, Jeyaseelan L, Peter JV, Cherian AM showed that for every 10 mEq/L reduction in serum sodium, patients were 17 times ( $p = 0.001$ ; confidence interval: 4-87) at risk of developing hiccups. The number of patients who had hyponatraemia with varying severity of hiccups showed a dose-response relationship. (PMID: 8664818[PubMed - indexed for MEDLINE] Hyponatraemia and hiccups.)



A study showed that there is a relationship between obstinate hiccup and hypocalcemia in patients after acute cerebral traumatic surgery. The concentrations of serum calcium were detected in 23 patients with obstinate hiccup, and 30 patients randomly selected without obstinate hiccup as controls. The concentrations of serum calcium in patients with obstinate hiccup were lower than in patients without obstinate hiccup. The difference of serum calcium in two groups was significant ( $P < 0.05$ ) which proves that the development of obstinate hiccup in patients after acute cerebral traumatic surgery is closely related to hypocalcemia. (Correlation between obstinate hiccup and hypocalcemia in patients after acute cerebral traumatic surgery(cba:613450) Qin Jun , Xu Daomiao , Ai Yuhang Department of Anesthesiology, Xiangya Hospital, Central South University.Changsha 410008,Hunan;China China Journal of Modern Medicine [2005, 15(2):289-290])

9 Aug 2015

The relation of praanaadi vaata bheda with Praana vahaanaam srotas ; iti praana sangyaka vaata vahaanaam . Etat cha \*praanaakya\* vishishtasya vaayoh vishishta srotah, saamaanyena tu vaayoh sarvaa eva dhamanya iti na virodhah.. it means it carries specifically praana vaayu.. Hetu ; kshayaat \* ( dhaatu kshaya or yakshmaa ) sandhaaranaat roukshyaat vyaayaamaat kshudhitasya cha → vaata prakopa ( specificaly praana vaayu ) → praana vaahini dushyanti → atisrishtam kupitam abhiksham sashabdam ( deep and rapid ventillation /hyperventillation/kussmaul's breathing as in metabolic acidosis ,etc ), atibaddham kupitam alpa alpam sashoolam ( shallow , slow breathing with pleuritic chest pain as in pleurisy/restricted chest diseases). These features are concerned with impairment of CNS , Airways , lungs parenchyma , and pulmonary circulation..chikitsa; shvaasiki chikitsaa ;as per shvaasa roga.. it means shvaasa roga is primary diseases of praanavaha srotas...now vaayu bheda description ; praana apaanau ; uchchhvaasa nihshvaasu..shvaasa karma is reffered as karma of praana vaata. Whether nihshwasa and uchchhvaasa both or only nihshvaasa , a



debate is welcome here. Praana apaanaau iti uchchhvaasa nihshwaasau , kechit tu praana apaanaau yathaa uktau eva vaatau praahuh ; tatra apaano yadyapi merdh shroni aadi aashraya eva iti aahuh , tathaapi \* hridayaavyatirikta anuvidhaayitvaat "hridaya aashrita " iti uchyate; referring uchchhvaasa karma by apaana since expiration helps in rid off undue CO2 ; end product of metabolism ( dhaatu mala ). After referring annam aadaana karmaa tu praanah koshtam prakarshayati , it seems as inspiration helps the entry of gases , karma of praana vaayu.. now applied approach ; vishvaga vrajati iti sarvato gachchhati ( discarded ventilation ) is due to yadaa srotaansi sanroodhya maarootah kaphapoorvakah sanroddhah tadaa and results in shvaasa roga. It shows mismatching between ventilation and perfusion , a leading pathophysiology of respiratory dyspnea.. urdhvam dhooyamaana vaatah (hyperventilation ) ,deergham shvasiti iti shvaasasya bahirnigamanam deergha kaalam karooti ( delayed forceful expiration ie FEV1sec is reduced ) , shvasiti vichchhinam iti nihshvasya punah kshana anten shvasiti ( cheyne stokes breathing & ataxic breathing) , n vaa shvasiti na shvaasam labhate ( apnea)etc; indicate impairment all components of respiratory system including cns and thoracopulmonary and pulmonary circulation.. only in aamaashaya gata vaata shvaasa roga is reffered , it shows the involvement of mahaasrotas by prakupita vaat. In praanaavrita udaana nihshvaasa uchchhvaasa sangrah ; shows interplay between praana and udaana ( both are urah sthaanashtha vaata bheda , so vulnerable to influence each other , if becomes prakupita ) chikitsa; urdhvabhaagikam karma /vamaanaadi . Udaanaa vrita apaana manifests shvaasadi roga( shvaasa hikka and kaasa ); pratilomam apaana vaayuh ( forceful expiration as in tamaka shvaasa : chikitsa- anulomanam , tamaku tu virechanam ) / udaana bhaavam aapannaa iti urdhvagati \*svabhaavam\*aapanah ( forceful or explosive expiration is cause of kaasanaat kaasah as in kaasa ). Kaphaavrita praana manifests nihshvaasa uchchhvaasa sangrah . In kaasa udaana and apaana , in shvaasa roga praana udaana and apaana are being affected.. After studying acharya sharangadhara ' s interpretation and all these references concerned with praanavaha sroto dushti ; the role of praana in nihshvaasa and

apaana in uchchhvaasa seems logic and clinically as well as therapeutically acceptable.. being sthaanashtha udaana vaata helps in respiration as a whole .After understanding the role of Cardiopulmonary circulation in perfusion it shows the role of vyaana vaata in rasa vikshepana. When vyaana vaata becomes abnormal as in pulmonary hypertension dyspnea manifests.. in metabolic acidosis and alkalosis hyper and hypo ventilation manifests respectively , the role of samaana vaata cant be ignored.... i think the role of panchaatmaa vaata is crucial in praanavaha srotas to maintain matching between ventilation and perfusion.. abnormality of any one leads to mismatching , a cardinal pathophysiology of shvaasa roga..There is inspiratory and expiratory center in medulla under control of praana and apaana respectively.. eg. In hypercapnea hyperventilation is to expell (nihsarana/utsarjana ) excess CO<sub>2</sub> , waste product \*(mala ) of carbohydrate and fat metabolism

13 Nov 2012

kaasaat shvaas kshayah chhardi svarsadaadayo gadah.bhavanti upekshaya..... AH.N.3/38 modern science;chronic bronchitis-COPD-(dyspnoea.).chr.bronchitis-pulmonary hypertension (haemoptysis) - Rt heart failure(systemic oedema)..+ sec.infection- pulmonary tuberculosis..laryngeal tuberculosis...etc...

20 Oct 2012

in pratamak shvas jvar,murchchhadi occur, means ,in patient of bronchial asthma, secondary infection occurs which aggravates airway obstruction and leads to severe hypoxia.santamak shvas is respiratory failure due to bronchial asthma...acharya chakrapani; pitta sambandhat jvaraadi yogen tamaksyaiv pratamak sangyam darshayannah...ch.chi.17/63-64..

11 September 2012

Tamakah kaph kase tu syat cha it pittanubandhajah. pittakaskriyam tatra yatha avastham prayojayet ch.chi.18/131. chakrapani; tamakah kas upadrav rupah.. when secondary infection occurs in patients of chronic Brochitis/COPD , clinically resembles with bronchial asthama, t/t antimicrobial, bronchodilators.

7 September 2012

Pratighat visheshen tasya vayoh saranhasah. vedana shabd vaishishtayam kasanamupajayate.ch.chi.18/9 chakrapani;pratighat iti aavaranam,sa cha kaphadi. e.g.in chronic bronchitis productive cough and coarse crepitations,in pneumonia cough,dyspnoea.chest pain etc with increased TVF, dullness on percussion,bronchial breathing,crepts,pleural rub ,etc.in both diseases pratighat vishesh manifests specific features.

27 August 2012

vishvagvrajati sarvato gachchhati;chakrapani on ch.chi.17/45.... means discarded ventilation which leads to mismatching between ventilation and perfusion and in turn hypoxia e.g. Emphysema (COPD)...

5 March 2014

**Obstructive sleep apnea/hypopnea syndrome ( OSAHS )** is defined as the coexistence of unexplained excessive daytime sleepiness with atleast five obstructed breathing events (hypopnea or apnea) per hour of sleep.. Obesity , hypothyroidism , Acromegaly , smoking are common cause.. C/F are daytime somnolence , impaired vigilance, cognitive performance and driving; depression; disturbed sleep , loud snoring and hypertension ;myocardial infarction ; stroke ; DM ; steatihepatitis.. T/t Continuous positive airway pressure , mandibular repositioning splint , surgery . Unfortunately , no drugs are clinically useful. Ayurved perspective ; t/t as per ati sthaulya - medoghna , pramehaghn , yoga.



14 SEP 2015

### **Acharya chakrapani on kloma ;**

Kloma hridayastha pipaasaa sthaanam ( acharya chakrapani on ch.vi.5/8), kloma pipaasaa sthaanam ( acharya chakrapani on ch.sh. 7/10) , kloma hridaya avayava visheshah ( acharya chakrapani on ch.chi. 13/45). These references show that kloma is a regulatory organ of ambuvaha srotas, to maintain fluidity in rasaraktaadi ambu dhaatu , 10 dooshya of prameha are enriched in aap mahaabhoota and need to have due amount of udaka for their transportation. Osmoreceptors in thirst center of hypothalamus regulate the amount of fluidity in blood, its activation is based on osmolality of plasma. Increased osmolality increases desire to intake of water, vice versa.. maarootah klomni samsthitah..... vardhayetam tadeva ambu svasthaanat udaraaya tau ( ch.chi 13/46) , svasthaana means peritoneal cavity which contains serous fluid similar to interstitial fluid.. there is exchange of fluid between plasma and interstitial space , dependent on hydrostatic and colloid oncotic pressure.. the imbalance can lead to accumulation of fluid in interstitial space /serous cavity and in turn edema/ascites/pleural effusion etc manifest.. this is reason why acharya charak describes udara chikitsa after shotha chikitsa.. intracellular and extracellular fluid maintainance is based on concentration gradient as well pressure gradient.. over all the maintainance of fluidity in body is well controlled by various factors, e.g. osmoreceptors, brain and atrial natriuretic peptides , Renin angiotensin aldosterone system.. osmolality of plasma is main initiator of activation of thirst center followed by others.. the balance between parthiva and aapa mahaabhoota in rasaraktaadi ambu dhaatu is essential to continue nonstop transportation.. the imbalance leads to sanga or atipravriti like sroto dushti.. the role of ambuvaha srotas is very critical to connect all cells with each other for their nutrition. Acharya chakrapani ; aahaara rasaat sarva dhaatu poshako dhaatu rasa utpadyate , sa cha \* raso dehaposhako ambu bhava \* iti aapya iti arthah . Tasya kshayaat iti rasa kshayat" trishyate", rasa kshayat ambu kshayo bhavati , ten cha ambu kshayena purushah "paaniya



prathanaa roopa" ( desire to intake of water ) trishnayaa yukto bhavati iti yuktam iti darshayati. Uktam hi sushrute - dosha dhaatu mala ksheeno bala ksheeno api maanavah , sva yoni vardhanam yat tat annapaanam prakaamkshati ( su.soo.15).. iti : ihaapi cha uktam tasya 'kshayaat cha trishyeddhhi 'iti ( on ch.chi. 22/16).all these references show the importance of ambu , pipaasaa and in turn pipaasaa sthaanam iti kloma; hridayastha pipaasaa sthaanam iti thirst center..Rasa roopo dhaatuh , kimvaam rasati iti raso drava dhaatuh uchyate , ten roodhiraadeenaamapi dravaanaam grahanam bhavati (acharya chakrapani on ch.chi 15/36)..

15 March 2015

**Trishnaamaandya can be correlated with adipsic hypernatraemia** ; a defect in the thirst mechanism results in adipsic hypernatraemia, a syndrome characterized by chronic or recurrent hypertonic dehydration. Deficient thirst is usually due to hypogenesis or destruction of the osmoreceptors in the anterior hypothalamus.it should be treated by administering water orally if the patient is alert and cooperative or by using hypotonic fluids (0.45% saline or 5% dextrose and water) via IV if the patient is not..

Acharya chakrapani ; kloma hridayastha pipaasaa sthaanam ( osmoreceptors ) is destroyed so there is imbalance between apa and prithvi dhaatu and manifests features in term of hypovolemia and hypernatraemia ; tachycardia , postural hypotension, azotemia, hyperuricemia( acharya charak mentions vaatarakta due to ati lavana ), and hypokalaemia .muscle weakness , pain , rhabdomyolysis, hyperglycaemia , hyperlipidaemia and acute renal failure may also occur. Apa dhaatu vridhhi is line of treatment so prithvi dhaatvaansha becomes normal relatively..Similar symptoms visible if sea water is drunk...due to hypernatraemia..

Many sailors caught in sea after the ship accident drink sea water directly & eventually die of dehydration & ARF...

सागराम्भस्त्रिदोषकृत्...

Visram tridosham lavanam ambu yat varunaalayam ( acharya chakrapani  
varunaalaye samudre , Visram ; aamagandhi ) ch.su. 27..

10 March 2016

## Cancer

Cancer is an exception to the coordinated interaction among cells and organs. In general, the cells of a multicellular organism are programmed for collaboration. Many diseases occur because the specialized cells fail to perform their assigned task. Cancer takes this malfunction one step further. Not only is there a failure of the cancer cell to maintain its specialized function, but it also strikes out on its own; the cancer cell competes to survive using natural mutability and natural selection to seek advantage over normal cells in a recapitulation of evolution. One consequence of the traitorous behavior of cancer cells is that the patient feels betrayed by his or her body. The cancer patient feels that he or she, and not just a body part, is diseased.

Carcinogenesis is not simply an event but a process, a continuum of discrete cellular changes over time resulting in more autonomous cellular processes. Prevention concerns the identification and manipulation of the genetic, biologic, and environmental factors in the causal pathway of cancer.

Cancer is the second leading cause of death behind heart disease. Deaths from heart disease have declined 45% in the United States. Once the diagnosis of cancer is made, the management of the patient is best undertaken as a multidisciplinary collaboration among the primary care physician, medical-oncologists, surgical oncologists, radiation oncologists, oncology nurse specialists, pharmacists, social workers, rehabilitation medicine specialists, and a number of other consulting professionals working closely with each other and with the patient and family. Because cancer therapies are toxic, patient management involves addressing complications of the disease and its treatment as well as the complex psychosocial problems associated with cancer. In the short term during a course of curative therapy, the patient's functional status may decline. Treatment-induced toxicity is less acceptable if the goal of therapy is palliation.

Further economically Cancer diagnosis, care and treatment is a great burden to the family and inturn to the society. The hospital stay and the surgical and adjuvant therapy cost is very high to bear by the common people which is also a cause for incomplete treatment procedure. Thus role of complimentary therapy holds the role in Cancer treatment to prevent progression of disease and also to prevent the complication of adjuvant therapy. Various herbal immune boosters are well known in Ayurveda which also have anti cancerous activity. It is the need of the hour to understand such drugs and their utility be studied in various types of Cancer.

20 March 2016

**वातोल्बणकामला-शाखाश्रित-त्रिदोषज-**

**(Cholangiocarcinoma -CCC )**

Mucin producing Adenocarcinoma of bile duct - रक्तवहस्रोतोसंगः,

Cirrhosis of liver (वातज-रौक्ष्य-धातुक्षय-काठिन्य-लाघव)

(rogaartikarshana ) - vaata prakopa ( tantra yantra dharah , kartaa garbhaakritinaam ) - change in cellular character -

Adenocarcinoma (वात-कफ-समूर्च्छितः)

अवरोधः शाखाश्रितकामला-

TREATMENT PLANNING

As per-

शाखाश्रित,कामला-



रक्तवहस्रोतस्,

पित्तज गुल्मः

1. रसोनः is bile secretagogue , it increases bile secretion ( vridhdya vishandanyaat..) So pitta will increase and will try to clear avarodha.. Rason also works on vaata and kapha directly , so , can clear the obstruction by action on Adenocarcinoma..

2. शरपुंखा

3. रोहीतकः

Both work on yakrita . For shaakhaa to koshta gati , rason will work. Vriddhi of pitta is accepted till aapittaranjana purisha , thereafter it will be stopped..

Here avarana is by vaata & kapha (?) , ideally it's sanga pradhaan ( kapha sammooorchchhita vaayu ) , and in turn aashaayapakarsha of pitta are consequences..

In phalashruti of rason ksheeram , vidradhi is indicated.. Cancer is utsedha pradhaana vyaadhi , so , it can be considered as one of shotha ,or gulma , or vidradhi .. Biliary tree is shaakhaa of yakrita , so , drugs indicated in yakrittodara , can be planned , here, in CCC..

4. भल्लातकः

गुल्मनाशनः ,:कफ हर-पित्त-वात ,छेदि(Antiobstructive) विबन्धहरः ,भेदनम् ,

5. ताम्रः

पित्तनिस्सारकःआयुष्यम् ,यकृत्प्लीहोदरहरः ,लेखनः ,

20 March 2016

**Ayurveda perspective of Cholangiocarcinoma ( CCC )** ; Mucin producing Adenocarcinoma of bile duct ( raktavaha srotasa ) , cirrhosis of liver ( rogaartikarshana ) - vaata prakopa ( tantra yantra dharah , kartaa garbhaakritinaam ) - change in cellular character - Adenocarcinoma.- ( vaata kapha sammoorchchhita ) - avarodha - shaakhaashrita kamala..treatment planning as per shaakhaashrita kamala , rakta vaha srotas ..

Even pittaja gulma should be considered .. Rason is bile secretagogue , it increases bile secretion ( vriddhya vishandanyaata..) So pitta will increase and will try to clear avarodha.. Rason also works on vaata and kapha directly , so , can clear the obstruction by action on Adenocarcinoma.. Sharapunkha rohitaka will work on yakrita . For shaakhaa to koshttha gati , rason will work. Vriddhi of pitta is accepted till aapittaranjana purisha , thereafter it will be stopped..

Here avarana is by vaata & kapha (?) , ideally it's sanga pradhaan ( kapha sammoorchchhita vaayu ) , and in turn aashaayapakarsha of pitta are consequences..

In phalashruti of rason ksheeram , vidradhi is indicated.. Cancer is utsedha pradhaana vyaadhi , so , it can be considered as one of shotha ,or gulma , or vidradhi .. Biliary tree is shaakhaa of yakrita , so , drugs indicated in yakrittodara , can be planned , here, in CCC..

#### CHOLANGIOCARCINOMA (CCC)

CCC typically refers to mucin-producing adenocarcinomas that arise from the bile ducts. They are grouped by their anatomic site of origin as intrahepatic, hilar (central, ~65% of CCCs), and peripheral (or distal, ~30% of CCCs).

They arise on the basis of cirrhosis, excepting primary biliary cirrhosis.

## **Aetiology**

Although most CCCs have no obvious cause, several predisposing factors have been identified, including primary sclerosing cholangitis, an autoimmune disease (10–20% of PSC patients), and liver fluke in Asians, especially *Opisthorchis viverrini* and *Clonorchis sinensis*.

CCC seems also to be associated with any cause of chronic biliary inflammation and injury, with alcoholic liver disease, choledocholithiasis, choledochal cysts (10%), and Caroli's disease.

## **Clinical Features:**

CCC most typically presents as painless jaundice, often with pruritus or weight loss, and acholic stools.

## **Investigation:**

Diagnosis is made by biopsy, percutaneously for peripheral liver lesions or, more commonly, via endoscopic retrograde cholangiopancreatography (ERCP) under direct vision for central lesions.

The tumors often stain positively for cytokeratins 7, 8, and 19 and negatively for cytokeratin 20.

However, histology alone cannot usually distinguish CCC from metastases from primary tumors of the colon or pancreas.

Serologic tumor markers appear to be nonspecific, but CEA, CA 19-9, and CA-125 are often elevated in CCC patients and are useful for following response to therapy.

Radiologic evaluation typically starts with ultrasound, which is useful in visualizing dilated

bile ducts, and then proceeds with either MRI or magnetic resonance cholangiopancreatography (MRCP) or helical CT scans.

Invasive ERCP is then needed to define the biliary tree and obtain a biopsy or is needed therapeutically to decompress an obstructed biliary tree with internal stent placement.

If that fails, then percutaneous biliary drainage will be needed, with the biliary drainage flowing into an external bag.

Central tumors often invade the porta hepatis, and locoregional lymph node involvement by tumor is frequent.

### **GALLBLADDER CANCER** (GB Ca)

GB Ca has an even worse prognosis than CCC, with typical survival of ~6 months or less. Women are affected much more commonly than men (4:1), unlike in HCC or CCC, and GB Ca is more common than

CCC. Most patients have a history of gallstones, but very few patients with gallstones develop GB Ca (~0.2%). It presents similarly to CCC and is often diagnosed unexpectedly during gallstone or cholecystitis surgery.

#### **Clinical Features:**

Presentation is typically that of chronic cholecystitis,

Chronic right upper quadrant pain and

weight loss.



Useful but nonspecific serum markers include CEA and CA 19-9.

CT scans or MRCP typically reveal a gallbladder mass.

The mainstay of treatment is surgical, either simple or radical cholecystectomy for stages I or II disease, respectively.

### **CARCINOMA OF THE AMPULLA OF VATER**

This tumor arises within 2 cm of the distal end of the common bile duct, and is mainly (90%) an adenocarcinoma. Locoregional lymph nodes are commonly involved (50%), and the liver is the most frequent

site for metastases.

Clinical Presentation:

Jaundice, and

many patients also have pruritus,

weight loss, and

epigastric pain.

Investigation:

Initial evaluation is performed with an abdominal ultrasound to assess vascular involvement, biliary dilatation, and liver lesions.

This is followed by a CT scan, or MRI and especially MRCP.

Adjuvant chemotherapy or radiotherapy has not been shown to be useful in enhancing survival. For metastatic tumors, chemotherapy is currently experimental.

Ayurveda can be better option in Cholangiocarcinoma (CCC), Carcinoma of the Ampulla of Vater and Gallbladder Cancer (Gb Ca). Following drugs may be used:

Lashuna, (*Allium sativum*) Garlic

Nimba, (*Azadirachta indica*), Neem tree

Sharpunkha, (*Tephrosia purpurea*)

Kumari, (*Aloe barbadensis*), Common Indian Aloe

Bhringaraj (*Eclipta alba*)

Ayurveda is better option as main line therapy or as complimentary therapy in various complications related to Cancer. Herbs mentioned above are just a bucket from the ocean. A good research in the field will help to improve the life of cancer patient without any economic burden.

19 March 2016

## **BLADDER CANCER**

Bladder cancer is the fourth most common cancer in men and the thirteenth in women.

### **EPIDEMIOLOGY**

Cigarette smoking is believed to contribute to up to 50% of the diagnosed urothelial cancers in men and up to 40% in women.

The aniline dyes, the drugs phenacetin and chlornaphazine, and external beam radiation.

Chronic cyclophosphamide exposure may also increase risk, whereas vitamin A supplements appear to be protective.

Exposure to *Schistosoma haematobium*, a parasite found in many developing countries, is associated with an increase in both squamous and transitional cell carcinomas of the bladder.

### **Clinical Presentation**

Hematuria occurs in 80–90% of patients and often reflects exophytic tumors.

After hematuria, irritative symptoms are the next most common presentation, which may reflect in situ disease.

Obstruction of the ureters may cause flank pain.

Symptoms of metastatic disease are rarely the first presenting sign.

### **Diagnosis And Staging**

Once hematuria is documented, a urinary cytology, visualization of the urothelial tract by CT or intravenous pyelogram, and cystoscopy are recommended if no other etiology is found.

The endoscopic evaluation includes an examination under anesthesia to determine whether a palpable mass is present.

An intraoperative video is often recorded.

Ultrasonography, CT, and/or MRI may help to determine whether a tumor extends to perivesical fat (T3) and to document nodal spread.

Distant metastases are assessed by CT of the chest and abdomen

Selective catheterization and visualization of the upper tracts should be performed if the cytology is positive and no disease is visible in the bladder.

Screening asymptomatic individuals for hematuria increases the diagnosis of tumors at an early stage but has not been shown to prolong life.

In Ayurveda, Uttar basti may be administered with help of various oils or decoction.

Associated therapy with Lepa and following Internal Medicine can be used:

Snuhi, (*Euphorbia neriifolia*), Milk Hedge

Apamarga, (*Achyranthes aspera*), Pricky-Chaff flower

Varuna, (*Crataeva nurvala*)

Punarnava, (*Boerhaavia diffusa*), Hogweed

Gokshur, (*Tribulus terrestris*), Small caltrops

## **PROSTATE CANCER**

### Aetiological Factors

Environmental factors may play a role.

High consumption of dietary fats, such as - linoleic acid, or the polycyclic aromatic hydrocarbons that form when red meats are cooked is believed to increase risk.



Similar to breast cancer in Asian women, the risk of prostate cancer in Asian men increases when they move to Western environments.

Diagnostic Criteria is based on symptoms,

an abnormal DRE (digital rectal examination), the DRE focuses on prostate size and consistency and abnormalities. Carcinomas are characteristically hard, nodular, and irregular, while induration may be due to benign prostatic hypertrophy (BPH) or to calculi or tumor.

an elevated serum PSA.

The urologic history should focus on symptoms of outlet obstruction, continence, potency, or change in ejaculatory pattern.

Prostate Biopsy

**Following herbal drugs can be used internally:**

Pashanbheda, (*Saxifraga ligulata*)

Deodar, (*Cedrus deodara*), Himalayan cedar

Snuhi, (*Euphorbia neriifolia*), Milk Hedge

Punarnava, (*Boerhaavia diffusa*), Hogweed

Varun, (*Crataeva nurvala*)

Brihat Panchmula It is group of 5 herbal drugs viz; Bilwa, {(*Aegle marmelos*), Bengal Quince}, Gambhari, {(*Gmelina arborea*)}, Patala, (*Stereospermum suaveolans*), Agnimantha, (*Premna integrifolia*), Syonaka, (*Oroxylum*

19 March 2016

### **PANCREAS CANCER:**

Over 90% of pancreatic cancers are ductal adenocarcinomas of the exocrine pancreas. These tumors occur twice as frequently in the pancreatic head compared to the rest of the organ, and tend to be aggressive, often presenting when locally inoperable or after distal metastases

have occurred. Patients with pancreatic cancer have a poor prognosis, with a 5-year survival of only 5%.

### **Aetiology**

Cigarette smoking,

Obesity,

Nonhereditary chronic pancreatitis appears to be risk factors for the development of pancreatic cancer.

Less clear, and sometimes conflicting associations, have been observed for other environmental factors such as diet, coffee and alcohol consumption, previous partial gastrectomy or cholecystectomy, and *Helicobacter pylori*.

An epidemiologic association between diabetes mellitus and pancreatic cancer has also been demonstrated; however, it is uncertain if diabetes is a precedent of, or consequence of, pancreatic cancer.

## **CLINICAL FEATURES**

Common presenting features of pancreatic cancer include pain (present in >80% of patients with locally advanced or metastatic disease), When present, pain is often felt as a dull ache in the upper abdomen and may radiate to the back, and characteristically may improve upon leaning forward. It may initially be intermittent, and may worsen with meals.

Obstructive jaundice,

Weight loss, these patients may suffer from marked weight loss, which may result from a combination of anorexia, early satiety, malabsorption or diarrhea/steatorrhea.

Anorexia.

Patients with jaundice may also have pruritus, pale stools, and dark urine; they often have tumors in the pancreatic head, and tend to be diagnosed earlier and with earlier stage disease.

Other symptoms tend to be more insidious, so that in the absence of jaundice, the interval between onset and diagnosis can be prolonged.

Other less common presenting features include the diagnosis of glucose intolerance (particularly within 2 years of cancer diagnosis), previous pancreatitis, migratory superficial thrombophlebitis (Trousseau's syndrome), gastrointestinal hemorrhage from varices, and splenomegaly.

### **Physical Findings**

Patients with early disease may not have any significant abnormalities detectable on physical examination.

Jaundice may be a presenting feature in some; in these patients a palpable, nontender

gallbladder (Courvoisier's sign) may be palpated under the right costal margin.

Patients with more advanced disease may have an abdominal mass, hepatomegaly, splenomegaly, or ascites. The left supraclavicular lymph node (Virchow's node) may be involved with tumor, or widespread peritoneal disease may be palpable on rectal examination in the pouch of Douglas.

### **Imaging Studies:**

Ultrasound is often used as an initial investigation for patients with jaundice, or with less-specific symptoms such as upper abdominal discomfort, and is able to assess the

biliary tract, gall bladder, pancreas, and liver.

Computed tomography (CT) scanning is preferable to ultrasound even though it is more

costly, as it is less operator-dependent, more reproducible, and less susceptible to interference from intestinal gas.

Endoscopic retrograde cholangiopancreatography (ERCP) is also widely used in the diagnosis of pancreatic cancer, particularly when CT and ultrasound fail to show a mass lesion, and may reveal either stricture or obstruction in either the pancreatic or common bile duct.



Endoscopic ultrasound (EUS) may be useful in the diagnosis of small lesions (<2–3 cm in diameter) and, in some cases, for local staging as well as evaluating invasion of major vascular structures.

EUS-guided fine-needle aspiration may also be used to obtain cytology for confirming the diagnosis, particularly in patients with potentially operable disease (see below).

Magnetic resonance cholangiopancreatography (MRCP) may be better than CT for defining the anatomy of the pancreatic duct and biliary tree, being able to image the ducts both above and below a stricture.

Positron-emission tomography with 18F-fluoro-2deoxyglucose (FDG-PET) may be useful for excluding occult distal metastasis in patients with localized disease who are being worked up for surgery or in patients with unresectable localized disease being considered for chemoradiotherapy.

**Serum Markers** The most widely used serum marker in pancreatic cancer is cancer-associated antigen 19-9 (CA 19-9). It has a reported sensitivity and specificity of about 80–90%, and is suggestive, rather than confirmatory, of the diagnosis of pancreatic cancer.

In context of Ayurveda Panchkarma: (Purificative Procedure) like Virechan may be useful. Internally following drugs may be used:

Nimba, (Azadirachta indica), Neem tree

Deodar, (Cedrus deodara), Himalayan cedar

Danti, (Boliospermum montanum), Habbussala

Pippali, (Piper longum), long pepper

Kiratatikta, (Swertia chirata), Chireta

Chitraka, (*Plumbago zeylanica*), Ceylon Leadwort

16 March 2016

## **SKIN CANCER**

### Clinical Characteristics:

There are four types of cutaneous melanoma. In three of these—superficial spreading melanoma, lentigo maligna melanoma, and acral lentiginous melanoma—the lesion has a period of superficial (so called radial) growth during which it increases in size but does not penetrate deeply. It is during this period that the melanoma is most capable of being cured by surgical excision. The fourth type— nodular melanoma —does not have a recognizable radial growth phase and usually presents as a deeply invasive lesion, capable of early metastasis.

When tumors begin to penetrate deeply into the skin, they are in the so-called vertical growth phase. Melanomas with a radial growth phase are characterized by irregular and sometimes notched borders, variation in pigment pattern, and variation in color. An increase in size or change in color is noted by the patient in 70% of early lesions.

Bleeding, ulceration, and pain are late signs and are of little help in early recognition. Superficial spreading melanoma is the most frequent variant observed in the white population.

The back is the most common site for melanoma in men. In women, the back and the lower leg (from knee to ankle) are common sites.

Nodular melanomas are dark brown-black to blue-black nodules. Lentigo maligna melanoma is usually confined to chronically sun-damaged, sun-exposed sites (face, neck, back of hands) in older individuals. Acral lentiginous melanoma occurs on the palms, soles, nail beds, and mucous membranes.

While this type occurs in whites, it is most frequent (along with nodular melanoma) in blacks and East Asians.

A fifth type of melanoma, the desmoplastic melanoma, is recognized. This tumor type is associated with a fibrotic response to the tumor, neural invasion, and a higher tendency to local recurrence.

Occasionally, melanomas can be amelanotic, in which case the diagnosis is established histologically after biopsy of a new or changing skin nodule or because of a suspicion of a basal cell carcinoma. Sites appear to be the forearm and leg (excluding feet), while unfavorable sites include scalp, hands, feet, and mucous membranes.

In general, women with stage I or II disease have a better survival than men, perhaps in part because of earlier diagnosis; women frequently have melanomas on the lower leg, where self-recognition is more likely and prognosis is better.

Lymphadenectomy may control early regional disease. Liver, lung, bone, and brain are common sites of hematogenous spread, but unusual sites, such as the anterior chamber of the eye, may also be involved.

Biopsy: The recommended technique is an excisional biopsy, as that facilitates pathologic assessment of the lesion, permits accurate measurement of thickness if the lesion is melanoma, and constitutes treatment if the lesion is benign.

For large lesions or lesions on anatomic sites where excisional biopsy may not be feasible (such as the face, hands, or feet), an incisional biopsy through the most nodular or darkest area of the lesion is acceptable; this should include the vertical growth phase of the primary tumor, if present. Incisional biopsy does not appear to facilitate the spread of melanoma.

**In ayurveda one can opt for Panchkarma: (Purificative Procedure) such as:**

Vaman

Virechan

2. Associated therapy

Lepa

Snehan especially various abhyanga

3. Internal Medicine

Manjistha, (*Rubia cordifolia*), Indian madder

Haridra, (*Curcuma longa*), Turmeric

Daruharidra, (*Berberis species*), Indian berberry

Bakuchi, (*Psoralea corylifolia*), Malaya tea

Chakramarda, (*Cassia tora*), Fetid cassia

Nimba, (*Azadirachta indica*), Neem tree..

15 March 2016

### **Neoplasm of the Lung**

The incidence of lung cancer peaks between ages 55 and 65 years. Lung cancer accounts for 29% of all cancer deaths (31% in men, 26% in women). Lung cancer is responsible for more deaths in the United States each year than breast cancer, colon cancer, and prostate cancer combined; more women die each year of lung cancer than of breast cancer.



## **Aetiology**

Most lung cancers are caused by carcinogens and tumor promoters inhaled via cigarette smoking etc.

## **Clinical Features**

Although 5–15% of patients with lung cancer are identified while they are asymptomatic, usually as a result of a routine chest radiograph or through the use of screening CT scans.

Most patients present Central or endobronchial growth of the primary tumor which may cause cough, hemoptysis, wheeze and stridor, dyspnea, and postobstructive pneumonitis (fever and productive cough).

Peripheral growth of the primary tumor may cause pain from pleural or chest wall involvement, dyspnea on a restrictive basis, and symptoms of lung abscess resulting from tumor cavitation.

Regional spread of tumor in the thorax (by contiguous growth or by metastasis to regional lymph nodes) may cause tracheal obstruction, esophageal compression with dysphagia, recurrent laryngeal nerve paralysis with hoarseness, phrenic nerve paralysis with elevation of the hemidiaphragm and dyspnea, and sympathetic nerve paralysis with Horner's syndrome (enophthalmos, ptosis, miosis, and ipsilateral loss of sweating).

Malignant pleural effusion often leads to dyspnea.

Pancoast's (or superior sulcus tumor) syndrome results from local extension of a tumor growing in the apex of the lung with involvement of the eighth cervical and first and second thoracic nerves, with shoulder pain that characteristically radiates in the ulnar distribution of the arm, often with radiologic destruction of

the first and second ribs. Often Horner's syndrome and Pancoast's syndrome coexist.

Other problems of regional spread include superior vena cava syndrome from vascular obstruction; pericardial and cardiac extension with resultant tamponade, arrhythmia, or cardiac failure; lymphatic obstruction with resultant pleural effusion; and lymphangitic spread through the lungs with hypoxemia and dyspnea.

Common clinical problems related to metastatic lung cancer include:-

Brain metastases with headache, nausea, and neurologic deficits;

Bone metastases with pain and pathologic fractures;

Bone marrow invasion with cytopenias or leukoerythroblastosis;

Liver metastases causing liver dysfunction, biliary obstruction, anorexia, and pain;

Lymph node metastases in the supraclavicular region and occasionally in the axilla and groin;

Spinal cord compression syndromes from epidural or bone metastases.

Adrenal metastases are common but rarely cause adrenal insufficiency

### **Investigation:**

Chest x-ray

CT scan of chest and abdomen

CT or MRI scan of brain and radionuclide scan of bone if any finding suggests the presence of tumor metastasis in these organs

Tumor tissue can be obtained by a bronchial or transbronchial biopsy during fiberoptic bronchoscopy; by node biopsy during mediastinoscopy; from the

operative specimen at the time of definitive surgical resection; by percutaneous biopsy of an enlarged lymph node, soft tissue mass, lytic bone lesion, bone marrow, or pleural lesion; by fine-needle aspiration of thoracic or extrathoracic tumor masses using CT guidance; or from an adequate cell block obtained from a malignant pleural effusion.

**Ayurved treatment by Panchkarma:** (Purificative Procedure) especially Vaman is very useful. Associated therapy with following procedures may also be helpful.

Ura- basti

Lepa

**Internal Medicine may be used:**

Pushkarmula, (*Inula racemosa*), janjabilsami

Bharangi, (*Clerodendron serratum*)

Ashwagandha kshar (*Withania somnifera*), Winter cherry

Haridra, (*Curcuma longa*), Turmeric

Daruharidra, (*Berberis species*), Indian berberry

Bhallataka, (*Semecarpus anacardium*) Marking nut

Vansha, (*Bambusa arundinacia*), Bamboo manna

Shirish, (*Albizzia lebbeck*)

Pippali, (*Piper longum*), long pepper

Tulasi, (*Ocimum sanctum*), Holy basil

Brihat panchmula: It is group of 5 herbal drugs viz; Bilwa, {(Aegle marmelos), Bengal Quince}, Gambhari, {(Gmelina arborea)}, Patala, (Stereospermum suaveolans), Agnimantha, (Premna integrifolia), Syonaka, (Oroxylum indicum).

14 March 2016

### **Colo-Rectal Cancer**

Cancer of the large bowel is second only to lung cancer as a cause of cancer death in the United States: 153,760 new cases occurred in 2007, and 52,180 deaths were due to colorectal cancer. Colorectal cancer generally occurs in person's  $\geq 50$  years. Most polyps produce no symptoms and remain clinically undetected. Occult blood in the stool is found in  $<5\%$  of patients with polyps.

### **Aetiological Factors:**

**Animal Fats** One hypothesis is that the ingestion of animal fats found in red meats and processed meat leads to an increased proportion of anaerobes in the gut microflora, resulting in the conversion of normal bile acids into carcinogens. This provocative hypothesis is supported by several reports of increased amounts of fecal anaerobes in the stools of patients with colorectal cancer. Diets high in animal (but not vegetable) fats are also associated with high serum cholesterol, which is also associated with enhanced risk for the development of colorectal adenomas and carcinomas.

**Insulin Resistance** The large number of calories in "western" diets coupled with physical inactivity has been associated with a higher prevalence of obesity. Obese persons develop insulin resistance with increased circulating levels of insulin, leading to higher circulating concentrations of insulin-like growth factor type I



(IGF-I). This growth factor appears to stimulate proliferation of the intestinal mucosa.

**Hereditary Factors:** Up to 25% of patients with colorectal cancer have a family history of the disease, suggesting a hereditary predisposition.

**Inflammatory Bowel Disease:** Large-bowel cancer is increased in incidence in patients with long-standing inflammatory bowel disease (IBD). Cancers develop more commonly in patients with ulcerative colitis than in those with granulomatous colitis, but this impression may result in part from the occasional difficulty of differentiating these two conditions.

#### OTHER HIGH-RISK CONDITIONS

**Streptococcus bovis Bacteremia:** For unknown reasons, individuals who develop endocarditis or septicemia from this fecal bacterium have a high incidence of occult colorectal tumors and, possibly, upper gastrointestinal cancers as well. Endoscopic or radiographic screening appears advisable.

**Tobacco Use:** Cigarette smoking is linked to the development of colorectal adenomas, particularly after >35 years of tobacco use. No biologic explanation for this association has yet been proposed.

#### CLINICAL FEATURES

Presenting Symptoms may vary with the anatomic location of the tumor.

Since stool is relatively liquid as it passes through the ileocecal valve into the right colon, cancers arising in the cecum and ascending colon may become quite large

without resulting in any obstructive symptoms or noticeable alterations in bowel habits.

Lesions of the right colon commonly ulcerate, leading to chronic, insidious blood loss without a change in the appearance of the stool.

Consequently, patients with tumors of the ascending colon often present with symptoms such as fatigue, palpitations, and even angina pectoris and are found to have a hypochromic, microcytic anemia indicative of iron deficiency.

Since the cancer may bleed intermittently, a random fecal occult blood test may be negative. As a result, the unexplained presence of iron-deficiency anemia in any adult (with the possible exception of a premenopausal, multiparous woman) mandates a thorough endoscopic and/or radiographic visualization of the entire large bowel.

Since stool becomes more formed as it passes into the transverse and descending colon, tumors arising there tend to impede the passage of stool, resulting in the development of abdominal cramping, occasional obstruction, and even perforation.

Cancers arising in the rectosigmoid are often associated with hematochezia, tenesmus, and narrowing of the caliber of stool; anemia is an infrequent finding.

### **Investigation:**

Digital rectal examination

Proctosigmoidoscopy as a screening tool was based on the observation that 60% of early lesions are located in the rectosigmoid.

Flexible sigmoidoscopy.

Colonoscopy

Double-contrast barium enema. Radiographs of the abdomen often reveal characteristic annular, constricting lesions ("apple-core" or "napkin-ring").

Occult fecal blood testing

Regardless of the clinicopathologic stage, a preoperative elevation of the plasma carcinoembryonic antigen (CEA) level predicts eventual tumor recurrence.

Screening:

Some authorities favor measuring plasma CEA levels at 3-month intervals because of the sensitivity of this test as a marker for otherwise undetectable tumor recurrence. Subsequent endoscopic or radiographic surveillance of the large bowel, probably at triennial intervals, is indicated, since patients who have been cured of one colorectal cancer have a 3–5% probability of developing an additional bowel cancer during their lifetime and a >15% risk for the development of adenomatous polyps.

In Ayurved Panchkarma (Purificative Procedure) like Virechan (Medicine induced Diarrhea) and Basti (Medicated oil enema or Medicated decoction enema) is very useful. The Purificative procedures help to reduce the overgrowth of gut microbiome.

**Following herbs may be used for internal use:**

Bilwa, (Aegle marmelos), Bengal Quince

Lodhra, (Symplocos racemosa), Symplocos bark

Snuhi, (Euphorbia neriifolia), Milk Hedge

Arka, (Calotropis gigantea) Madar, Gigantic Swallow wort

Patha, (Cissampelos pareira), Velvet leaf

Manjistha, (*Rubia cordifolia*), Indian madder

Bakuchi, (*Psoralea corylifolia*), Malaya tea

Bhallataka, (*Semecarpus anacardium*) Marking nut

Nishoth, (*Operculina turpethum*), Turpeth root

**Especially for Rectum Cancer following drugs and procedure may be prescribed**

Chavya, (*Piper officinarum*)

Mochrasa (gum of *Salmalia malabarica*)

Piccha basti.

## **BREAST CANCER**

Breast cancer is a malignant proliferation of epithelial cells lining the ducts or lobules of the breast. Epithelial malignancies of the breast are the most common cause of cancer in women (excluding skin cancer), accounting for about one-third of all cancer in women.

### **Aetiology**

Increased caloric intake contributes to breast cancer risk in multiple ways: earlier menarche, later age at menopause, and increased postmenopausal estrogen concentrations reflecting enhanced aromatase activities in fatty tissues.

Moderate alcohol intake also increases the risk by an unknown mechanism. Folic acid supplementation appears to modify risk in women who use alcohol but is not additionally protective in abstainers.



Breast cancer is a hormone-dependent disease. Women without functioning ovaries who never receive estrogen-replacement therapy do not develop breast cancer.

### **Breast Examination**

Women should be strongly encouraged to examine their breasts monthly.

Breast examination by the physician should be performed in good light so as to see retractions of nipple and other skin changes.

The nipple and areolae should be inspected, and an attempt should be made to elicit nipple discharge.

All regional lymph node groups should be examined, and any lesions should be measured.

Physical examination alone cannot exclude malignancy.

Lesions with certain features are more likely to be cancerous (hard, irregular, tethered or fixed, or painless lesions).

A negative mammogram in the presence of a persistent lump in the breast does not exclude malignancy.

Palpable lesions require additional diagnostic procedures including biopsy.

In premenopausal women, lesions that are either equivocal or nonsuspicious on physical examination should be reexamined in 2–4 weeks, during the follicular phase of the menstrual cycle. Days 5–7 of the cycle are the best time for breast examination.

A dominant mass in a postmenopausal woman or a dominant mass that persists through a

menstrual cycle in a premenopausal woman should be aspirated by fine needle biopsy or referred to a surgeon.

If nonbloody fluid is aspirated, the diagnosis (cyst) and therapy have been accomplished together.

Solid lesions that are persistent, recurrent, complex, or bloody cysts require mammography and biopsy, although in selected patients the so-called triple diagnostic techniques (palpation, mammography, aspiration) can be used to avoid biopsy.

Ultrasound can be used in place of fine-needle aspiration to distinguish cysts from solid lesions.

Not all solid masses are detected by ultrasound; thus, a palpable mass that is not visualized on ultrasound must be presumed to be solid

Better mammographic technology, including digitized mammography, routine use of magnified views, and greater skill in mammographic interpretation, combined with newer

diagnostic techniques (MRI, magnetic resonance spectroscopy, positron emission tomography, etc.) may make it possible to identify breast cancers even more reliably and earlier. Screening by any technique other than mammography is not indicated; however, younger women who are BRCA-1 or BRCA-2 carriers may benefit from MRI screening where the higher sensitivity may outweigh the loss of specificity.

Not Recommended Test in Breast Cancer

Complete blood count

Serum chemistry studies

Chest radiographs

Bone scans

Ultrasound examination of the liver

Computed tomography of chest, abdomen, or pelvis

Tumor marker CA 15-3, CA 27-29

Tumor marker CEA

**Ayurvedic Treatment that can be recommended in breast cancer is as follows:**

Panchkarma: (Purificative Procedure) especially Vaman (Vomiting induced by medicine) will be very useful as a preventive as well as curative therapy.

Internal Medicines which have a role in Breast Cancer are as follows:

Vacha (*Acorus calamus*), Sweet Flag

Kutki (*Picrorrhiza kurroa*), *Picrorrhiza*

Shilajit (Bitumen),

Guduchi (*Tinospora cordifolia*), *Tinospora*

Bhallataka ghrit, (*Semecarpus anacardium*) Marking nut

External Application in the form of paste following drugs may be used:

Kombadnakhi ,

---

Shigru (Moringa pterygosperma), Drumstick tree.

11 May 2014

**Myeloid Sarcoma** is extramedullary leukemic tumor , with acute myeloid leukemia. Its myeloid neoplasm. It is found in skin , bone and lymph nodes. It also involves cns , oral and nasal mucosa, breast, genitourinary tract , chest wall , pleura, retroperitoneum , GIT and testes. C/F -compressive signs , severe pain and abnormal bleeding.. CT Scanning, MRI and biopsy are helpful for diagnosis. T/T-Chemotherapy ( cytarabine ) , Radiotherapy and Hematopoietic stem cell transplantation..In Ayurved perspective its Raktaj Arbud.. The drugs acting on rakt and majja dhatu may be effective..

2 July 2014

**Organo-sulfur compounds found in garlic** have been identified as effective in destroying the cells in glioblastoma, a type of deadly brain tumor. Diallyl sulfide ,a compound in garlic, is effective in intestinal infection caused by campylobacter bacterium. Garlic oil may help protect diabetes patients from cardiomyopathy.. Other uses are prostate cancer, lungs cancer, Hip osteoarthritis , common cold, bronchitis, cholelithiasis, etc...Lashuna(garlic)kshiram is indicated in vaata gulma, udaavartam, gridhrasim , vishama jvaram , hridrogam , vidradhim and shotham.. ch.chi.5/94-95..

Fenugreek or Methi are a rich source of constituents known as steroidal saponins that help to reduce the body's absorption of cholesterol coming in through the fat rich foods we eat. Saponins have a role to play in reducing the body's production of cholesterol.



Garlic contains sulphur compounds that act as antioxidants and also help with dilatation of the blood vessels , keeping blood pressure at normal level. It reduces LDL-Cholesterol and avoids deposition of plaque on arterial wall ,reducing chances of AMI, IHD and CVA..

### Posts on various other Topics

13 June 2015

Balya dravya are maansapushtikara, it means such dravya can be used in muscular atrophy with other dravya as per causative factors.. Balya dravya are guduchi , balaa, atibalaa, shaalmali, gokshura, bilva ,guggula, yashtimadhu, kapikachchhu, kooshmaanda, gandhaprasaari, saarivaa, shankhapushpi, ashwagandhaa, gambhaaree, rasona, and shataavari etc.. these have variant qualities eg few are ushna tikta , few are sheeta madhura , acting on agni and dhaatunirmithi respectively.. selection of dravya from above list depends upon whether initial factor is agni dushti or maansa dhaatuvaahi srotas or both.. in muscular dystrophy both are affected , while in myestheni gravis maansa dhaatuvaahi srotas is affected , in malabsorption syndrome ( grahani dosha ) agni dushti is initial factor.. maansadagdhataa is present in kaamala ; guduchi yashtimadhu kooshmaanda gambhaari shataavari like dravya are indicated which have tikta madhura sheeta qualities... In periarticular muscular atrophy ( OA ) ; guggula with kapikachchhu yashtimadhu gokshuru ashwagandhaa saarivaa can be used since these dravya have madhura guroo guna property..guggula being sandhaankrita help in healing of articular breeches so can prevent loss of calcium from articular cartilage.. this is one reason why guggula kalpa are indicated in sandhigata vaata etc.Above mentioned balya dravya can be used in proteinuria to supplement the loss; guggula gokshuru shataavari ashwagandhaa may be best choice..Approach to nighantu helps in selection of dravya in different conditions in different patients..

31 Dec 2014

Sanyaasa or coma ; Raktavaaheeni rasavaaheeni sangyaavaaheeni cha, prithak prithak samastaa vaa srotaansi kupitaa malaah..... sanyaasah teshaam.... (ch.su 24/25-27). Hepatic encephalopathy, hypertensive encephalopathy ,diabetic ketoacidosis coma , Hyperosmolar hyoperglycaemic state, uraemic coma myxedema coma etc are related to rakta and rasavaaheeni srotas which become rajamoha aavrita atmanah so impaired consciousness is found in these disorders.. epileptic seizures , hypercarbia , stroke etc are related to sangyaavaaheeni srotas.. basically all causes of sanyaas are initially related to rasa and rakta vaaheeni and later sangyaavaahi srotas is involved so person becomes \*kaashthibhooto mritopamah (vegetative state)\* .. t/t sadyah phalaah kriyaah-\*anjani avapeedah cha dhoomah pradamanaani cha. .. hinguoshana samaayuktam yaavat sangyaa prabodhanam \*\* (ch.su 24/44- 53). Emergency management is indicated with potent n prompt treatment.. such as virechan is helpful in hepatic encephalopathy , IV insulin in diabetic coma , IV antihypertensive in HTN encephalopathy , Haemodialysis in uraemia , raktamokshana in presence of increased intracranial pressure..etc..

25 Dec 2014

Milk/Curd proteins, particularly caseins have appropriate amino acids composition for growth n development of young. Other proteins in milk include an array of enzymes,proteins involved in transporting nutrients, protein involved in diseases resistance, growth factors etc. Caseins are highly digestible in intestine and high quality source of amino acids. Most whey proteins are relatively less digestible in intestine , although all of them are digested to some degree. When substantial whey proteins is not digested fully , some of intact protein may stimulate a localized intestinal or systemic immune response . This is , due to beta

lactoglobulin, referred to a milk protein allergy.. for preventing allergy dry ginger is added in milk and aamalaki/ honey in curd.. curd protein is best for strengthening muscles.. in most of charakokta tail mentioned in vaata vyaadhi contain milk n curd to make it effective in neuromuscular diseases.. curd never increase spasticity if taken day time and patient is mobile or doing vigorous exercise.. curd is recommended by neurophysicians even in UMN diseases .. It has nothing to do inflammation or any reaction other than allergy/intolerance..

It contains immunoglobulins to protect from infections/diseases.. Caseins are present in curd.. Dadhi is uhna amla vaataghna so it does not induce spasticity.. even in peenase sheetake vishama jvare dadhi is recommended achary charak ( ch.su 27/225-227)

8 April 2016

### **Lysosomal storage disease in ayurveda perspective ;**

the lysosome is commonly referred to as the cell's recycling center because it processes unwanted material into substances that the cell can utilize. Lysosomes break down this unwanted matter via enzymes, highly specialized proteins essential for survival. Lysosomal disorders are usually triggered when a particular enzyme exists in too small an amount or is missing altogether. When this happens, substances accumulate in the cell. In other words, when the lysosome does not function normally, excess products destined for breakdown and recycling are stored in the cell. It means there is defect in agni at cell level , therefore , aama is produced , which will work as aamavisha to cell.. For this purpose , katu, tikta , deepaniya, paachaniya dravya are best choice .. Pippali , rasona , chitraka , patola , kiratatikta , etc..With help of anulomana/virechan , we can clear excess products.. Haritaki like drugs should be added with deepaniya and paachaniya dravya. Mahasudarshan , lashunaadi vati , arogyavardhini vati , gandharva haritaki ,



vardhamaana pippali.. Trikatu , or panchakola shrita jala , and /or shadangapaaniya. Takraarishta . Samaana and apaana are major concern. In samaana aavrita apaana hridroga , grahani dosha are mentioned. Deepaniya ghrita is choice. Here , next choice is to see the condition as aamapradoshaja vikaara or aamavaata like conditions.. Multiple organ systems are involved.. So , treatment plan as per aamavaata.. Enzymes are paittika entities.. But , my concern is how enzymes become deficient ? I think that its because of samaanaavrita apaana leading to production of unwanted and that unwanted becomes accumulated in cell.. Therefore , we have to transform unwanted into wanted , for this we need katu and tikta . tikta is helpful to control the production of unwanted . and katu will help to convert unwanted into wanted . second , by using virechaniya dravya , elimination of unwanted material , if not transformed into upachita dhaatu . Vaayu + agni for transformation , and vaayu + aakaasha to maintain apratighaatatva , so, we can achieve normalcy in cell.. Deepaniya ghrita is best choice.. Vardhamaana pippali is an other choice.. बृंहणं स्वर्यमायुष्यं प्लीहोदरविनाशनम् । वयसः स्थापनं मेध्यं पिप्पलीनां रसायनम्..च.चि.1/3/40

4 April 2016

The balance of Eastern and Western knowledge :

Seed standouts: flax and hemp

Flax seeds are a tremendous asset to the vegetarian diet. They also have an interesting history. It is believed that flax and flax seeds were first cultivated in Babylon in 3000 B.C. Hippocrates used flax for patients with abdominal complaints, around 650 B.C. Around the eighth century, Charlemagne passed laws actually requiring people to add flax to their diets, because of how important he viewed flax to be to health (17). We are not required to eat flax seeds, but it sure is a good idea to do so! Flax seeds are among the best plant sources of omega-3 fats, plus they have lignans, an anti-carcinogen, and boron, a mineral important



for bone health. Best to eat them ground, so that the nutrients are readily available (the tiny seeds are easy to swallow whole). It's a no-brainer to add ground flax seeds to mixed dishes, hot cereal and smoothies. And if you need an egg replacer for cooking, blend 1 tablespoon ground flax with 3 tablespoons water.

Hemp seeds are another super source of omega-3 fatty acids, and are showing up everywhere these days - cereals, "milk," cookies and bars, and even vegan ice cream. The seeds (and their oil) offer the greatest health benefits.

Why not just use the oil?

Flax and hemp oils, as expected, contain more omega-3 fats per serving than the whole seed. So why not just skip them and go directly to the extracted oil? Actually, it's not a bad idea to use high omega-3 oils in moderation. But the oil should not replace the seeds; they should both be incorporated into the diet. The whole seeds contain fiber and other important nutrients that do not end up in the oil. But the oil has concentrated amounts of protective fats. So both are important. Oils high in omega-3s oils spoil rapidly and should be kept in the refrigerator and used within a few weeks. These oils are perfect for salad dressings and smoothies but, due to low smoke points, not suitable for cooking. Healthy vegans should aim for 1/2 to 1 teaspoon of flax or hemp oil a day, depending on the rest of the diet.

Bottom line

If you are a vegan and concerned about your health, nuts and seeds should play a role in your daily diet. Their nutrient profiles, not to mention their flavor and versatility, go a long way in making the optimal vegan diet as nutritious and delicious as it can be.

(1-17) References for this article are available from NAVS atnavs@telenet.net or P.O. Box 72, Dolgeville, NY 13329.

प्रमेही तथा अतसी सर्षप तैलयुक्तम् 20/6.चि.च ..

31 Oct 2014

### **Teeth Grinding (Bruxism)**

Approximately 15% to 33% of children grind their teeth. Children who grind their teeth tend to do so at two peak times -- when their baby teeth emerge and when their permanent teeth come in. Most children lose the teeth grinding habit after these two sets of teeth have come in more fully.

Most commonly, children grind their teeth during sleep rather than during waking hours. No one knows exactly why children grind their teeth but considerations include improperly aligned teeth or irregular contact between upper and lower teeth, illnesses and other medical conditions (such as nutritional deficiencies, pinworm, allergies, endocrine disorders), and psychological factors including anxiety and stress.

Grinding of the baby teeth rarely results in problems. However, teeth grinding can cause jaw pain, headaches, wear on the teeth, and Temporomandibular disorders.

Specific tips to help a child stop grinding his or her teeth include:

Decrease your child's stress, especially just before bed.

Try massage and stretching exercises to relax the muscles.

Make sure your child's diet includes plenty of water. Dehydration may be linked to teeth grinding.

Ask your dentist to monitor your child's teeth if he or she is a grinder....

**Drooling (also known as sialorrhea)** is the flow of saliva outside the mouth. Drooling can be caused by excess production of saliva, inability to retain saliva within the mouth (incontinence of saliva), or problems with swallowing (dysphagia or odynophagia).

Isolated drooling in healthy infants and toddlers is normal and is unlikely to be a sign of either disease or complications. It may be associated with teething. Drooling in infants and young children may be exacerbated by upper respiratory infections and nasal allergies.

Some people with drooling problems are at increased risk of inhaling saliva, food, or fluids into the lungs, mainly if drooling is secondary to a neurological problem. However, if the body's normal reflex mechanisms (such as gagging and coughing) are not impaired, this is not life-threatening.....Causes

Stroke and other neurological pathologies

Intellectual disability

Cerebral palsy

Drooling associated with fever or trouble swallowing may be a sign of an infectious disease including:

Retropharyngeal abscess

Peritonsillar abscess

Tonsillitis

Mononucleosis

Strep throat.....Treatment of patients with drooling problems has been successful at some centers using a team

approach, including an otolaryngologist, pediatric dentist, speech pathologist, and physical therapist.

Aggressive physical medicine or medical management prior to considering surgical intervention is recommended. Medical management is directed towards correcting the oral motor dysfunction and decreasing the secretory volume of salivary glands.

### **Thumb sucking**

At birth, a baby will reflexively suck any object placed in its mouth; this is the sucking reflex responsible for breastfeeding. From the very first time they engage in nutritive feeding, infants learn that the habit can not only provide valuable nourishment, but also a great deal of pleasure, comfort, and warmth. Whether from a mother, bottle, or pacifier, this behavior, over time, begins to become associated with a very strong, self-soothing, and pleasurable oral sensation. As the child grows older, and is eventually weaned off the nutritional sucking, they can either develop alternative means for receiving those same feelings of physical and emotional fulfillment, or they can continue experiencing those pleasantly soothing experiences by beginning to suck their thumbs or fingers. This reflex disappears at about 4 months of age; thumb sucking is not purely an instinctive behavior and therefore can last much longer. Moreover, ultrasound scans have revealed that thumb sucking can start before birth, as early as 15 weeks from conception; whether this behavior is voluntary or due to random movements of the fetus in the womb is not conclusively known.

Thumb sucking generally stops by the age of 5 years. Some older children will retain the habit, which can cause severe dental problems. While most Dentists would recommend breaking the habit as early as possible, it has been shown that



as long as the habit is broken before the onset of permanent teeth, at around 5 years old, the damage is reversible. Thumb sucking is sometimes retained into adulthood and may be due to stereotypic movement disorder, another psychiatric disorder, or simply habit continuation

23 Sept 2014

**Triad ( 3 )** ; Tremors, rigidity , Bradykinesia- Parkinson's disease.. Dyspnea, orthopnea and paroxysmal nocturnal dyspnea - left heart failure. Raised JVP , Congestive hepatomegaly and edema - Right heart failure... Edema , hypertension and proteinuria & hematuria - glomerulonephritis.. Proteinuria , hypoproteinaemia and edema - nephrotic syndrome.. abdominal pain , hematuria and hypertension - Renal artery thrombosis..

12 SEP 2014

**Duchenne's muscular dystrophy (DMD)**; the dystrophin -glycoprotein complex appears to confer stability to the sarcolemma. Primary deficiency of dystrophin may lead to secondary loss of sarcoglycans and dystroglycan. Disruption of dystrophin - glycoprotein complexes weakens sarcolemma, causing membrane tears and a cascade of events leading to muscle fibre necrosis.. this sequence of events occurs repeatedly during life of a patient with muscular dystrophy.. DMD may benefit from either replace defective gene or missing protein ( dystrophin) or implement downstream corrections (e.g. skipping mutated exons)..ayurveda; maansa is matrija bhaava ie x-chromosomal disease.. in DMD deficiency of maansaagni is present so saara bhaaga(dystrophin-glycoprotein complex) is not produced and leading to riktaani mansavaha srotaansi (muscle necrosis) poorayitvaa anilo bali(ch.chi28/18) maansagata vaata and manifests gurvangam..... shramitam ati artham ( undue fatigue).. etc..(ch.chi28/32)..

t/t vireko maansamedahsthe niruhaa shamanaani cha (ch.chi 28/93) so we have to think about drugs acting on maansaagni , "saamaanyam vridhi kaaranam " for replacement of specific maansa dhatvaansha...shaman and niruha for both maansa and vaata..

Basically dhatu nirmaana is being affected so there is less saara bhaaga utpatti and in turn weakening of dhaatu ie necrosis..since there is rikta srotas due to necrosis so vaata prakopa occura to fullfill srotas and in turn there is pseudohypertrophy.. so i think initially there is maansa gata vaata , later due to pseudohypertrophy there is aavrita vyaana ie maansaavrita vyaana..t/t; vrihat vaata chintaamani rasa , laghumaalini vasant , pravaala panchaamrit rasa , combination of ashwagandha shataavari balaa kapikachchhu maasha saarivaa manjishthaa mustaa amritaa aamalaki haritaki vidanga daarvi bilva pippali... snehana ,pinda sveda, maansa rasa basti/yaapanaa basti , physiotherapy and hydrotherapy..

20 April 2013

Sarv bhootanam karanam akaranam satv rajastamo lakshanam asht roopam akhilasya jagatah sambhav hetuh avyaktam nam... Tadekam bahoonam kshetra gyanaamadhishtanam samudra(sea) evaudakanaam bhavanam... Su.Sh.1/3 Life on earth is due to biogenesis, from avyakt to ahankar, continuous growing biomechanisms , from prakriti to purush.. prakriti... beej dharmini prasav dharmini.... Su.Sh. 1/13....

4 August 2012

Amlo raso raktam dushayati.... Lavano rasah raktam vardhayati. katuko raso shonitsanghatam bhinatti.. charak sutrasthanam 26

17 August 2012

Acharya charak mentioned genetic factor as one of aetiology of obesity:chakrapani stated bij svabhavat iti sthoolmatapitrijanyatvat charak sutrasthan 21/4

5 May 2013

First time charak mentioned about closed circulation-> santatya bhojya dhatoonam parivrittistu chakravat.. ch.ch.15/21.acharya chakrapani ; bhojye upyukte sati dhatoonam rasadinam chakravat parivrittih bhavati avishranta samutpattih dhatoonam bhavati..

Vyanen ras dhaturhi vikshep uchit karmana. Yugpat sarvato ajasram dehe vikshipyate sada. Ch.ch.15/36.. Acharya Chakrapani ; ath ko annrasam raktadi dhatu poshakam prerayati; yen tatra tatra rasah sarpati iti ah-vyanen ityadi.. Rasroopo dhatuh , kinvam rasat iti raso drav dhatuh uchyate, ten roodhiradinam api grahanam bhavati; vikshepah uchitam prakritam karma yasya s vikshep uchit karmah; ten vyanen, yugpat iti ek kalam, sarvat iti sarvasmin dehe, vikshipyate iti niyate, ajasram iti avishrantam vikshipyate, sadeti sarv kalam... Best explanation of human circulation(close circulation) by acharya charak and acharya chakrapani.....

1 April 2013

On charak sidhisthan-9/4 acharya chakrapani stated that although the place of apan is medhradi,but it may be even hriday,because-hridayaavyatiriktaanuvidhayitvat hridayaashrit uchyate.. Hriday plays a role in circulation of rasaadi ambu dhatu,which constitute not only dhatu sarbhag but also kittabhag,through vyan vat karm.. vyanen rasdhaturhi vikshepochitkarmana..ch.chi15/36- but as apan is responsible to eliminate kitta,it is apan which transports the kitta to be excreted through orifices.. This is reason why acharya charak mentions hridgadah in saman avrit apan(ch.ch.28/205

13 April 2013

Tatra abhighatje vayuh prayo raktam pradhushyan. Sa vyatha shoph vaivarnyam karoti sa rujam jvaram. Ch. Chi 3/113-114 Cardinal signs

( vyatha,shoph,vaivarnyam,ruja,jvar)

The classic signs and symptoms of acute inflammation:

English Latin \*Redness-Rubor \*Swelling-Tumor \*Heat-Calor \*Pain-Dolor \*Loss of function-Functio laesa...

Trauma-vayu prakop- rakt dushti - Inflammation.

18 March 2013

SAhaasyahaasi sanmuhyan praledhi dashanachchhadau. Shitpaadkarochchhvaaso yo naro na sa jeevati.. ch.ind.11/20.. shock is defined by the presence of multisystem end-organ hypoperfusioon.clinical indicators include reduced mean arterial pressure,tachycardia,tachypnea, cool skin and extremities,acute altered mental status,and oliguria. The end result of multiorgan hypoperfusion is tissue hypoxia,often clinically manifested by lactic acidosis..

19 March 2013

Sanubadhanam cha dirghkaalaavasthaayikushthaadivikaarkaari.. acharya chakrapani on ch. chi.1/1/5.. delayed type of hypersensitivity(DTH) reaction is tissue damaging response in leprosy and tuberculosis which develops about 2 to 4 weeks after infection..

1 April 2013

On charak sidhistan-9/4 acharya chakrapani stated that although the place of apan is medhradi,but it may be even hriday,because-



hridayaavyatiriktaanuvidhayitvat hridayaashrit uchyate.. Hriday plays a role in circulation of rasaadi ambu dhatu, which constitute not only dhatu sarbhag but also kittabhag, through vyan vat karm.. vyanen rasdhaturhi vikshepochitkarmana..ch.chi15/36- but as apan is responsible to eliminate kitta, it is apan which transports the kitta to be excreted through orifices.. This is reason why acharya charak mentions hridgadah in saman avrit apan(ch.ch.28/205

5 May 2013

The plasma binding proteins increase the pool of circulating hormones, delay hormones clearance, and may modulate hormones delivery to selected tissue sites... Dhatu poshak rasvahi vyan roopah..(chakrapani on ch.su.28/3).. One of function of vyan vayu is through transporters, plasma-binding proteins.. perfusion to tissue as per metabolic demand is also function of vyan vayu-> vyanen ras dhaturhi vikshep uchit karmana.ch.chi.15/36.. Hypoperfusion to tissue (ischaemie) is due to decreased action of vyan vayu...!

2 May 2013

Rakt kshayo raktposhak rasasya pitten kshapanad rakt poshak sar bhag (iron,Vit B12, folic acid etc) anutpaadaat ( deficiency) cha...chakrapani on ch.chi.16/6...

1 May 2013

Snigdha-ushna ; urdhvag-kaph sansrishtam -virechan- tarpanadi kramo hitah... Ushna-ruksham ;adhog-marutaanugam- vaman-yavagvadi.. ch.ch.4...urdhvag-oroward peristalsis-virechan-anoward peristalsis; counter mechanism... Adhog-anoward peristalsis-vaman-oroward movement-counter mechanism... pratimargam (counter mechanism) cha haranam raktpitte vidhiyate..ch.ni2/19..

12 April 2013

Jvara pratyatmikam lingam \*(sine qua non - derived from latin word- sine kwa: 'no:n)\* santapo deh manasah.ch.chi.3/31.. acharya chakrapani- atm lingam iti avyabhichari lakshanam- indispensable and essential action, condition or without which \*( there is)\* nothing..

10 August 2012

Sven tenoshmana chaiva kritva dehoshmano balam ch.ch.3/130 chakrapani stated sveneti dehoshmanah; teneti jathargnyushmana.dehoshman iti sakaldehcharinah prakritoshmanah means increased core temperature.

15 Aug 2014

Ebola virus disease (EVD) is also known as ebola hemorrhagic fever. Incubation period is 1 wk.. contagious in nature , hospital acquired infections.. early features are fever rashes headache nausea vomiting abd.pain low backache arthritis sore throat. later ; bleeding from mouth ear nose. Genital swelling rashes all overbody conjunctivitis.etc.. pneumonia and liver failure are complication. Prognosis is bad , fatal disease. CBC , Coagulation time Bleeding time liver function test.. Ayurved ; Rapid happening of disease indicates its sannipattika nature.. in charak chi 3/103-109, some features of sama sannipaata jvara are similar to EVD ; ashti sandhi shiroruja , sasaraave kalushe rakte nirbhugne chaapi darshane.. shthivanam raktapittasya , kothaanaama syaavaraktaanaama mandalaanama chaa darshanama.. chikitsa ; kaphasthaanaanupoorvyaa vaa sannipaata jvaram jayet.Drugs; patola , nimba , kantakari , vaasaa , kiratatikta , amrita , saptaparni , shataavari, durvaa , svarna sootashekhara rasa etc.

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Prakshepa drugs like pippali , maricha, shunthi etc are bioenhancers or biopotentiators, are the agent capable of enhancing bioavailability and efficacy of

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a drug with which its co-administered , without any pharmacological activity of its own at therapeutic dose used. A fixed drug combination (Resorine for TB) of rifampicine , isoniazid and piperine contains 60% less than of rifampicine because of its increased bioavailability and it also prevents resistance. Piper nigrum extracts enhanced activity of pefloxacin, norfloxacin , ampicillin, diclofenac sodium , carbamazepine , phenytoin ,propranolol , metronidazole etc. The prakshep drugs also have synergistic action proved by researchers.. The best ideology of our great Acharya...Quercetin, found in citrus fruits , is bioenhancer and also exhibits biological activity including antioxidants, radical scavengers , antiinflammatory , antiatherosclerosis , antitumor , and antiviral , etc.. ( vayahasthaapanaanaam)

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Chromosomal aneuploidies ; यदा त्वस्याः शोणिते गर्भाशयबीजभागावयवः स्त्रीकराणां च शरीरबीजभागानामेकदेशः प्रदोषमापद्यते , स्त्र्याकृतिभूयिष्ठां अस्त्रियं ( असंपूर्णस्त्रीलक्षणम्( वार्ता ( female hypogonadism, Turner's syndrome ) . यदा त्वस्य बीजे बीजभागावयवः पुरुषकराणां च शरीरबीजभागानामेकदेशः प्रदोषमापद्यते , तदा पुरुषाकृतिभूयिष्ठं अपुरुषं ( असमस्तपुरुषलक्षणयुक्तं ) तृणपुत्रिकं ( male hypogonadism, Klinefelter ' s syndrome ) नाम जनयति.. च.शा. 4/30-31..

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## CASE STUDIES

Ayurveda for Atrial fibrillation(AF).. Kamala kshaara as anticoagulant.. Prabhaakara vati and combination of arjuna shatavari brahmi to control rate..The action of Vana plaandu is similar to digoxin (+ ionotropic)..

1 March 2015

Neuropathy; Here some ayurveda treatment based on clinical experiences of senior consultants of ayurveda including me.. one can select as per severity and chronicity of peripheral neuropathy ; local application by laghupanchamoola or dashamoola + erandamoola + devadaru + pushkaramoola and shunthi.. vishatinduka tail/sahachara tail abhyanga. Indication of chandrakala ras is in antharbahir dahascha, which is one of the main sign of early neuropathy.

vishatinduka vati, ekangavira rasa , yogaraja guggula, kvaath of dashamoola ashwagandha erandamoola rasna chopachini amrita amalaki devadaru vijayasar shunthi n brahmi vati.. Neuropathy ;Demyelination ( kapha kshaya ) - vaata vridhhi - pittasya aashaayapakarsha - burning sensation...Mallagarbha pottali, pravalgarbha pottali with dashamoola kwatha, indu vati, vishamusti vati, punarnavaadi mandoora.

If confirm as demyelination disorder then give abragarbha pottali/abhraka bhasma with varunadi kwatha along with above combination. just try adding a little lashauna to any oil of your choice, potentiate it and apply locally. Swarnabhupathi ras 150 mg twice . Ksheera bala 101 /Dhanwantaram 101 20 drops with milk , masha tailam abhyanga is excellent in burning ,which is chief complaint in neuropathy, will gradually decrease.



26 Aug 2015

Case report ; On 20/8/15, a female aged 30 yrs came with complaints of abdominal pain, bilaterally at both flanks , nausea.. pain was severe , colicky type at both renal angle regions. On examination ; 74/min , 130/80, CVS ; normal , RS ; normal , P/A ; tenderness on both renal angle .diagnosis ; vrikakashmari. USG revealed 5 X 3 mm calculus in upper pole calyx of Lt kidney and 4 X 3 mm calculus in middle pole calyx of rt kidney..urinalysis revealed few crystals otherwise normal . i started chandraprabhavati 500 mg tds , gokshuraadi guggula 500 mg tds , shankha vati 250 mg tds , kvatha of punarnava gukshuru varuna paashaanabheda shataavari and haritaki ( each 2 gm ), 50 ml bd. 3-4 lt water intake /day..I advised diclofenac 2 cc , IM , stat , later there was no need of any antispasmodic drug , since abdominal pain did not recur.. On 26/8/15 , i recommended follow-up USG , it revealed 4 X 3 mm calculus in mid pole calyx of Lt kidney.. calculus in rt kidney is removed out , and of Lt kidney has been decreased in size and shifted lower from upper pole calyx to mid pole calyx..

27 Aug 2015

Case report ; female patient ,aged 21 years, came , with Lt ear pain , purulent discharge with offensive smelling since 1 month (known case of tympanic membrane perforation with otitis media ) , in late evening of 12 aug -15 , commonly i never see the patient of shalya shaalaakyaadi (paraadhikaara ) deptt , but patient came in late evening after routine opd time , so i had to handle properly.H/O modern treatment with antimicrobials, analgesics. on examination i found tympanic perforation and purulent discharge. S/E - normal. As per ayurveda perspective , krimi -→pitta prakopa & rakta dushti.. i prescribed gandhak

rasayan vati , sookshma trifala vati , arogyavardhini vati , and combination of khadira yashti saarivaa manjishtha kushtha nimba patola vidanga tulasi ( each 500 mg ) vachaa trikatu pravala panchaamrita ( each 250 mg ), bid.. yesterday she came again in late evening with no earache , no discharge. On examination , i found clear external ear canal , little decrease in circumference of perforation. I continued the same for an other 7 days and told her to come in morning opd time to consult shaalaakya tantra tagya for confirmation about hearing acuity and perforation size..

27 Aug

Case report ; One month back i admitted male patient aged 35 yrs with complaints of loss of appetite, heart burn , abdominal pain , abdominal distension , giddiness , breathlessness on exertion , and edema. H/O alcohol consumption since 15 yrs , about 180 ml /day. Belongs to poor socioeconomic status. On examination ; icterus , HR-90/min , week , regular , BP - 110/70 mm of Hg. JVP -normal. CVS: S1S2 normal , no added sounds. RS ; normal vesicular bronchial breathing sound. P/A - tendered hepatomegaly (2 fingers ) , splenomegaly ( 3 fingers ) , bulged flanks , fluid thrill + ve, shifting dullness , traversed umbilicus , dilated veins at flanks of abdomen.. CNS - oriented , conscious. Pitting pedal edema.. Dx; yakridodara with jalodara .. lab ; Abnormal LFT , sr bilirubin 12 mg %. Hb - 6 gm % , USG ; Hepatomegaly , PHT , splenomegaly , ascites. Samprapti ; madya →pitta prakopa →amlapitta →raktavaha srotodushti →yakridodara→maarutah klomni sansthitah ( portal hypertension) →srotahsu rooddhamaargeshu kaphah cha udaka moorchchhitah ( transudation and lymphatic obstruction ) →vardhayetam tat eva ambu svasthaanaat udaraaya tau ( increased fluid in peritoneal cavity ) →udaka udaram iti →jalodara ( ascites).

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PHT →splenic venous congestion ; vimaargagamana →pleehodara... chikitsa sootra ; hetu parivarjana ; abandoned alcohol , drugs acting on pitta ,vaata , rakta , yakrita and pleeha. Mridu Virechana per day. Mootrala dravya. Drugs given ; chandraprabha vati , gokshuradi guggul, punarnavadi mandoor , mahasudarshana ghan vati , phalatrikaadi with rohitaka sharapunkha punarnava and pippali as kvaath , gandharva haritaki vati , erandra patra svarasa.. on examination ; no icterus , liver n spleen are not palpable, no fluid thrill , no abdominal distension , no edema. Lab ; normal liver function .sr bilirubin 1.7mg %, Hb 9 gm %. Follow up USG is recommended. He is still in IPD..

23 Sept 2015

Today , one female ,married, school teacher , aged 30 yrs , came for the purpose to reduce weight.. her weight ; 71 kg , height ;1.58 meter, BMI; 28.4..small frame..systemic exam is normal.. mala, mootra , jivhaa, nidraadi are normal.. on counselling i found she and her family are consuming salt in pohe/uppita, chapati , rice , varana/aamati, daala, sabji/bhaaji , chatani of ground nuts , atleast 10gm/day/individual.. edible oil;2kg/month/individual.. no fruits in diet.. milk is there atleast 250 ml/day.. i adviced to stop mixing salt in chapaati and rice..and to decrease in sabji daala etc, it should not be more than 5 gm/ day..To consume oil 500 gm/individual/month.. total calory intake sould not be more than 1000 calory/day.for calculation of calory i gave her chart consisting of quantity with calory of each and every used diet..to start walking atleast 4-5 km/hr for all days of week.. i prescribed kaanchanaara guggula 1gm bid, trifalaa guggula 500 mg bid , kvaatha of combination of trifalaa, mustaa ,vidanga, amritaa , bilva(each 2 gm), haridraa , daaruharidraa , kustha , devadaaru and trikatu ( each 1gm ), 75 ml bid .. after 1 week follow up..

sama maansa pramaanastu sama sanhanano narah , dhridhendriya vikaaraanaam na balenaabhibhooyate. Kshutpipaasaa aatapa saha sheeta vyaayaama



sansahah.sama paktaa samajarah samamaansa chayo matah.. ch.soo..21/18;19.. these are features of prashasta purusham.. upachaya is desirable.. atiupachaya ; atisthaulya and alpa upachaya;atikrisha .. maansa shabdena eha upachayo vivakshitah ..Refer hetu ; these are concerned with increased intake of calory with decreased expenditure of calory.. genetic predisposition ; beeja svabhaavaat iti sthoolamaataapitrijanyatvaat.. medasvina iti hetu garbha visheshanam (e.g overweight baby of diabetec mother ). Everything is mentioned in charak samhita : need to explore..please refer here acharya chakrapani; ten yasmaat atisthoole shareere medo dehavyaapakatven labdhavriti , atastadeva praayo vardhate naanye rasaadayah , tadabhibhootatvaadi iti arthah..Our genetic predisposition leads to typical indian abdominal belly ; make us vulnerable for type2 DM.. therefore, i consider atisthaulya as metabolic syndrome/X syndrome..Madhu is best example of guru apatarpanam cha.. here , i am impressed with European population , they prefer honey in place of sugar..Since centuries we are indulged in high santarpanotha aahaara and vihaara.. acharya charak was very much consciuos about diet as svaasthya paraka and vikaara paraka.. after astaunindita adhyaaya , he discussed langhana brinhaniya adhyaaya..

4 Feb 2016

Psoriasis ( ekakushtha, kitibha ) ; Since few months , I am treating the cases of psoriasis with the combination of trifala bakuchi manjistha nimba vidanga saarivaa tulasi patola each 500mg bid , with water, orally. External application by Lepa of the combination of nimba sariva manjistha khadira kushtha daarvi haridra bakuchi , each in same quantity and as per need , mixed well with gomootra , followed by sun exposure for 20 mins. The result starts within a week therapy , after a month , itching , scaling are decreased significantly , there is good improvement in discoloration , after 3 months skin becomes almost normal , then I ask to stop application , and to keep continue drugs orally for an other month. No panchakarm ..

10 Feb 2016



In cases of vipaadikaa , I use combination of trifala daarvi khadir haridra nimba patola yashtimadhu saarivaa manjistha and kushtha mixed with water to make paste for local application , twice in a day . orally gandhaka Rasayan vati 250 mg bid , kamadudha 250 mg bid.. Today , my one elder female patient of vipaadikaa came on follow up , after 15 days treatment.. Almost all fissures in skin of both feet have been healed , slight roughness of skin is remained.. She had severe fissures with sometimes bleeding , and unable to walk due to pain since 1 year.. She was treated by too many physicians (dermatologist too ) , but disease was progressive .. I observed that bad hygiene , not caring feet , bare feet walking , agriculture work etc are common causes of vipaadikaa in rural population. Taking care of local hygiene is need to explain to each and all patients of vipaadikaa.. Soaps are also found as one of cause , in those cases soaps especially clothe washing soaps must be avoided.. I randomly select the cases of vipaadikaa for local application either with gandhaka malahara , or sarjarasa malahara , or above combination . I find the above mentioned combination is better than these two malahara.. In few cases I used above mentioned combination and gandhaka malahara both separately to apply twice a day ( total 4 times ) , result is better , but inconvenience to apply four times restrict the patients to continue the same on all days..it's curable disease , subject to patient's cooperation to follow the instructions..

27 Feb 2016.

Case report ; Elder female , CKD , HTN , DM , urine output less 400 ml per day , blood urea - 96 mg % , SR. Creatinine 4.9 mg % on 4/2/16 , with higher side BSL and border line Hypertension. Admitted in College hospital and managed by chandraprabha ,gokshuraadi guggula , shveta parpati , punarnavadi mandoora , kvaatha of dashamoola punarnava varuna pashanabheda kantakaari sariva manjistha Arjun shatavari amrita bilva musta, mahasudarshan ghan vati.. Today , blood urea 49 mg % , Sr. Creatinine 1.9 mg % FBSL 98 mg % . , urine output , since

one week , 1200 - 1700 ml /day.. BP is absolutely normal.. Clinically well settled..Before 6 month , she was diagnosed with CKD. HTN and DM , since more than 15 years..

9 March 2016

Case report ; Yesterday , young male aged 20 years came with palpitation , chest pain , headache , giddiness and fatigue , on examination ,HR ; 120/min , BP ; 170/100 mm of Hg , JVP ; normal , CVS ; Loud S1S2, no murmurs , no any added sounds. RS ; Clear , P/A normal, no organomegaly , no bruits on renal arteries.. No edema , normal peripheral pulsations. I prescribed ; prabhakar vati 125 mg bid , combination of Arjuna, shatavari , brahmi, kamala, pushkaramoola , devadaru , punarnava, gokshuru , each 500 mg , shunthi , and pravaala panchaamrita , each 250 mg , ( 4.5 gm ) , bid , with water .. I did ECG ; Sinus tachycardia , otherwise normal. I advice for 2-D colour Doppler echocardiogram.. Just , patient came , no palpitation, no chest pain , no headache , no giddiness , no fatigue , feeling of well-beingness. Echo revealed normal report.. On examination ; HR ; 76/min , BP ; 120/76 mm of Hg.. No sarpagandha ghan vati , no modern drugs. Just , after 3 doses , patient is about normal.. I told him to come on tomorrow for follow up , the same , above mentioned , drugs are continued. Here, temperature is max 30'. No issue of sun stroke , I examined thoroughly to confirm secondary causes , and advised 2D echo.. Everything is normal.. No stress , no exam time.. At this level , I think it as essential Hypertension.. Increased heart rate and in turn increased cardiac output leading to Hypertension.. Sympathetic overstimulation may be reason , but no underlying cause is observed.. At this level , I am not thinking about pheochromocytoma , however , I advice USG & color Doppler -abdomen to rule out renal and adrenal causes of Sec. Hypertension. ..

16 March 2016

Case report ; on 14th march , elder male aged 56 years came in OPD with low backache , tingling and numbness in both lower extremities , constipation ( passage of hard stool in alternate days ), headache . non hypertensive , non diabetic , non alcoholic , non smoker . on examination ; HR -76/min , BP -190/110, JVP0 ,CVS - S1normal S2 loud , no added sounds , RS normal , P/A normal , SLR : + ve bilaterally , knee , ankles and plantar reflexes are diminished. ECG is done , normal findings . BSL is normal , CBC -normal , urinalys - normal. X-ray ; lumbosacral AP & Lateral view , degenerative changes with osteophytes ; lumbar spondylosis.. Rx- sinhadad guggul 500 mg bid , lakshadi guggula 500 mg bid , Vishatinduka vati 250 mg bid , gandharva haritaki vati 4gm at night , combination of punarnava gokshuru vacha jatamansi shankhapushpi each 1gm , tds , snahana svedana and Kati basti.. Admission in morning time .. In evening BP was 180/100 , so , I decided not to start modern antihypertensive drug.. On 15th in morning BP was 170 /100, in evening 160/100, today in morning 140/90 mm of Hg. Now no headache , bowel motion is normal. There is improvement in low backache and numbness & tingling, SLR is normal.. my approach to patient is as per ashthi majja gata vaata / guda gata vaata & Hypertension.. If , we consider the transit time is concerned with constipation ( as in guda gata vaata and purishaavrita vaata ) , we have to give drugs acting as snigdha anulomaka to facilitate normal bowel motion either in morning time or at night or at both times. Different procedures are adviced and would be done in day time , so , soft bowel motion in morning is more applicable and desirable.. I find , after giving gandharva haritaki at night , there is help by lying down posture to provide better efficacy of gandharva haritaki or any anulomaka dravya..

26 April 2016

Yesterday , one middle aged female came with गलग्रह (dysphagia ) , on examination , it's observed that she is suffering from severe anaemia , after test ,Hb% is found 5.2 GM % , microcytic hypochromic RBC.. The combination of symptomatic proximal Esophageal webs ( cause of dysphagia ) and iron deficiency



anemia in middle - aged women constitutes Plummer - Vinson syndrome ; iron deficiency , angular stomatitis , glossitis , and dysphagia ( raises the risk of oral squamous cell cancer and Esophageal cancer at the postcricoid tissue web ). See, the observation of acharya charak ; हृत्पाण्डुग्रहणीरोगप्लीहानाहगलग्रहान् कासं कफजमर्शांसि यावशूको व्यपोहति..च.सू.27/305 ; yāvashūka is indicated in both pāndu and galagraha.. I prescribed it with tāpyādi lauha. I told her to consult oncologist for further evaluation of disease and management , but she is too poor to afford. There is no angular stomatitis and glossitis , therefore , I am not much worried , so , I started treatment.. Let's pray for her recovery.. My thinking behind using yavakshaara is to work on web , if present.. And web is kaphaja in nature.. यस्य श्लेष्मा प्रकुपितः तिष्ठति अन्तर्गले स्थिरः . आशु संजनयेत् शोफं जायते अस्य गलग्रहः । च.सू.18/22..

23 April 2016

Adult female , aged 34 years , Shopkeeper , came , one week before , with low backache , numbness and tingling in both legs , difficulty in walking , discomfort while sitting and standing since 5 years. 10 hours working in her own shop. On examination ; HR ; 74/min , regular .BP ; 120/80 mm of Hg.. CVS ,RS , P/A normal. Pain on palpation over L-3-4-5 level.. SLR + ve.. Knee jerks , ankle jerks are diminished.. X- Ray - lumbosacral AP & lateral view revealed lumbar spondylosis with degeneration and osteophytes.. Diagnosis ; ashthimajaagata vaata .. Mechanical loading - vaata prakopa - ashthi majja gata kha vaigunya - ashthimajaagata vaata.. Treatment given ; mahavatvidhvansha rasa 125 mg bid , yogaraj guggul 500 mg TDS , lakshadi guggula 500 mg TDS , Vishatinduka vati 250 mg bid , combination of ashwagandha bala shatavari chopachini each 1 GM + godanti and shunthi each 250 mg , bid with milk. Snehan, svedan, and Kati basti.. After 7 days of treatment , now significant reduction in all symptoms , SLR is



negative.. Important information about numbness and tingling , which became very less..

21 April 2016

I admitted one male elder with IHD on yesterday and one female elder with IHD on today.. With ST - segment and T - wave changes , similar oral treatment as given to anaesthetist , posted few days back on Facebook , with hrida basti by tila taila for 20 mins .. Male patient have multiple coronary artery disease with 95% block of RCA , and 70% of 3 branches of LCA.. He is adviced for CABG , but , because of his financial status , he can't afford the charges , so , he came to me to seek the help of Ayurveda.. An other female patient came with fever , burning micturition , frequent urination and chest pain.. Known case of Hypertension.. ECG revealed ST - segment and T- wave changes in inferior and apical wall of myocardium.. First time , this female patient is diagnosed with IHD at our hospital.. We started similar management for IHD to both patients.. Later , I will post details about response to our treatment .. Our college is at Kodoli kolhapur , rural area. Our continued efforts with population now could develop trust in ayurveda.. Therefore , the patients with disease related with any organ system come to our hospital , even for emergencies ! The work is accountable for making society aware of health and ayurveda..!

14 April 2016

Case report ; young male , professionally anaesthetic , from Haryana , known case of IHD , symptomatic , with ST - segment and T- wave changes in ECG.. Distance Medicine , I told him to take ; Arjun shatavari pushkaramoola punarnava devadaru gokshura haritaki amalaki kamala tulasi ( each 500 mg ) + bilva chitraka

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trikatu vacha ( each 250 mg ) / day in 2 divided doses with honey.. Rason ksheeram at night. Tulasi leaves wet ginger and black pepper in tea.. After 15 days of treatment , his ECG became normal. His consulting cardiologist also appreciated the result.. Treatment plan as per vaatika hridroga with avarana as hetu and special consideration of samaana and apaana , saama meda , kapha for dhamanipraticchaya.

5 April 2016

Case report ; female , adult , came with severe pain in right leg below knee joints 20 days back. No previous history of any major illness. On examination ; varicosities of Rt superficial saphenous vein , hotness , tenderness , redness and cord like palpation over vein courses.. S/E normal.. No neurological findings.. सिरागत वात ( निदानार्थकर हेतु ) - रक्तावृत्त वात.. Thrombophlebitis.. Drugs ; chandraprabha vati 500 mg bid , gokshuraadi guggul 500 mg bid , mahavatvidhvansha rasa 250 mg bid , arogyavardhini vati 250 mg bid , kvaath of combination ; dashamoola erandamoola haritaki kamala yashtimadhu amrita punarnava varuna pashanabheda each 1 GM shunthi 500 mg , 40 ml bid . lepa by dashanga dashamoola kamal yashtimadhu amalaki haritaki punarnava mustaa each 2 GM , bid .. After 10 days , on first follow up , there was good response , but pain and other features were present.. Today , on 2nd follow up ; no pain , no tenderness , no hotness , no redness. Just varicosities are remained.. Same treatment is continued with mahavatvidhvansha rasa 125 mg bid.. No contemporary Medicine intervention , no panchakarm , no physiotherapy. Considering vaata , rakta , sthaanika shotha and Raktaavrita vaata , treatment plan as per Raktaavrita vaata and in turn vaatarakta .. Vaataghna , raktaprasaadaka , mootrala , shothaghna.. Sanghaatabhinnaatikara dravya were selected.



..to be continued...